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## PREFACE TO ELEVENTH EDITION

It has been an onerous task revising this book for its eleventh edition. Dr Roxburgh was not only a brilliant teacher but his approach to dermatology was essentially practical, and these two great virtues are reflected on every page of his text. Although the whole work has been carefully reviewed, the majority of it remains unchanged. Where alterations or additions have been made, every effort has been taken to emphasise the main purposes of the book which are to endeavour to give lucid descriptions and explanations for the medical student and straightforward therapeutic directions for the general practitioner.

New paragraphs have been added dealing with streptomyces eruptions, purpura dermatitis including that caused by carbromal, cross-sensitisation, trichophyton rubrum infection, nickel dermatitis, Kaposi's varicelliform eruption, senile haemangioma and skin plating. Major alterations have been made in the sections dealing with the general use of drugs including antibiotics and steroid hormones, varicose veins, impetigo, lupus erythematosus, rosacea, fungous infections of the hands and feet, the treatment of tuberculosis of the skin and of dermatitis herpetiformis and the use of ultra violet rays. Minor alterations have been made where appropriate.

The chapter on syphilis has been entirely re-written and re-illustrated. The section on seborrhoeic eruptions has been moved from the chapter on diseases due to vegetable parasites to that on diseases of the sebaceous and sweat glands.

One of the major attributes of the book has always been the illustrations. Apart from those dealing with syphilis, only seven have been replaced and eleven have been added.

The practice of making occasional bibliographic references has not been continued although those mentioned in the tenth edition, if still appropriate, have been retained. It was considered

that a full bibliography is out of place in a book of this type and that a partial one can be misleading

It is a pleasure to thank Dr R M B MacKenna for his constant interest, help and encouragement. I am also greatly indebted to Dr O S Nicol for the new chapter on syphilis to Mr G W Taylor for advice on skin affections due to cold and to Miss T Wareham for the section dealing with the uses of ultra violet rays. For the illustrations I have to thank Mr N K. Harrison and his Staff of the Photographic Department, and Dr I M P Goode of the Skin Department, St. Bartholomew's Hospital. The photograph of Kaposi's varicelliform eruption was taken for me by Dr David Cogman and is reproduced by kind permission of the Editor of the *Lancet*

PETER BORRIE

115a HARLEY STREET  
LONDON W1  
June 1956

## PREFACE TO FIRST EDITION

WHEN Messrs. H. K. Lewis suggested that I should write them a book on Skin Diseases for their General Practice Series I at first demurred because there are already many excellent text-books of all sizes and degrees of completeness. They pointed out, however that a complete treatise was not required that only the common diseases need be considered and that these should be described from the point of view of diagnosis and such treatment as the general practitioner is in a position to give. After discussing the matter with colleagues I agreed to try and I should like here to acknowledge my indebtedness to my friend and colleague Professor George E. Gask, who has throughout stimulated me to find time to write this book.

I should like to emphasize the fact that this is not a complete text book, as it only deals with some one hundred and twenty out of more than three hundred skin diseases which are known. The difficulty throughout has been to deal adequately and readably with the common skin diseases without making the book unduly large and for this reason no account is included of the anatomy and physiology of the skin nor of congenital affections, the bullous eruptions, the erythrodermias, the atrophies scleroderma,<sup>1</sup> pigmentary anomalies, nor of the less common diseases in the classes which are described, nor of the treatment of syphilis, which is fully described in books on venereal diseases. Neither is any account given of the technique of electrical X ray or Radium treatment, as it is improbable that general practitioners will use these themselves. The indications for such treatments and the results to be expected are, however fully dealt with. Ultra violet light and carbon dioxide snow which are used by many practitioners, are described in more detail.

Accounts of these conditions were inserted in the second edition.



Stress has been laid throughout on differential diagnosis, for experience has shown me that it is here that practitioners and students go astray when confronted with skin eruptions. The space devoted to differential diagnosis has materially increased the size of the book, but I believe it is space well used.

To assist the student an index of preliminary diagnosis has been placed at the beginning of the book to encourage him to observe accurately the type of lesion of which any given eruption is composed, and to guide him as to what diseases to look up in this book or in larger works.

Some may think that an unduly large proportion of the book is given up in the first three chapters to general considerations of etiology, pathology, signs, symptoms, diagnosis and treatment, but it is my endeavour to get my students to approach dermatology as a rational subject and not as a mere collection of hybrid Greek and Latin names associated with more or less complicated prescriptions.

For any virtues the book may be found to possess I must express my indebtedness first to my friend and teacher Dr H. G. Adamson whose Chief Assistant I was privileged to be for ten years and to whom I owe my training in dermatology. Secondly to the inspiration of the late Professor Josef Kyrle of Vienna, whose classes I attended in 1935 less than a year before his untimely death robbed dermatology of one of its most distinguished exponents.

I must express my debt also to the text-books of MacLeod and Sequeira on which I have drawn freely and to the writings of Whitfield and Barber especially the latter's *Lettsomian Lectures* of 1929. For the histology of many conditions I have drawn upon McCarthy's *Histopathology of Skin Diseases*.

I am indebted to Dr Lindsay Batten for reading portions of the manuscript and criticising them from the general practitioner's point of view.

I must express my thanks also to Messrs. H. K. Lewis & Co. Ltd. who asked me to write this book and have done their best to meet my wishes in its production.

The illustrations are mostly from photographs from my own

cases at St. Bartholomew's or St. John's Hospitals. Where this is not so the source is stated, and I thank the donors for their kindness in lending me photographs.

I am indebted to the Authorities of the British Museum (Natural History) for permission to reproduce certain drawings of animal parasites from their Economic Series and to Dr MacLeod for the loan of the blocks of these drawings, and of Fig 48 which appeared in his text-book also to Dr O'Donovan and Messrs. Churchill for the loan of the block of Fig 47.

The coloured illustrations are from colour photographs by Colour Photographs Limited, the negatives being taken by Messrs. Navara.

I hope it is a fact of good omen that this small book emanates from the same address as Radcliffe-Crocker's famous text-book, viz. 121 Harley Street.

A. C. ROXBURGH

April, 1932.

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# INDEX OF PRELIMINARY DIAGNOSIS FOR STUDENTS

## How to use this Index

1 Read descriptions on pp 8 to 13 of the different types of lesions found in skin diseases, unless you already know them.

2 Ascertain by careful inspection the nature of the lesions of which the eruption under consideration is made up.

3 Select, from the diseases whose names are given under the type of lesion found, those which seem most probable. Refer to the descriptions of them which will be found at the pages indicated.

4 Make your final diagnosis on the points there given.

NOTE.—Diseases not followed by a page number are not described in this book. These are mostly either diseases dealt with in textbooks of medicine or surgery or diseases relatively unimportant or uncommon in Great Britain. For these reference must be made to other and larger works.

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Varicella.

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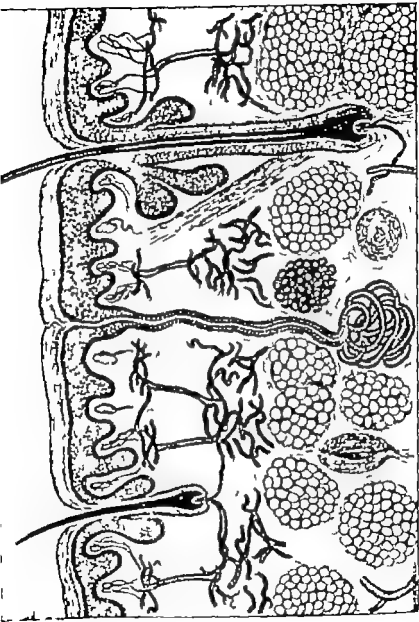


Diagram of Structure of Skin.

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# COMMON SKIN DISEASES

## CHAPTER I

### GENERAL ETIOLOGY AND PATHOLOGY

THE skin, being the part of the body which comes in contact with the outside world, is subject to injury by many agencies which do not affect the internal organs. It follows that there is a much greater variety in the causation of skin diseases than in that of visceral diseases. Moreover the skin being visible, slight differences in its response to injury or infection are easily recognized, whereas similar differences in the response of internal organs are not appreciable. Consequently the number of skin diseases which are known is very large, being over 300 if only the main varieties are considered.

Skin diseases are no exception to the general rule that before any given agency can produce a "disease" the patient must be susceptible to it. This fact must be borne in mind when considering the long list of possible causes of skin disease. The susceptibility of different individuals to injury by various agents is a matter of degree and may vary from time to time in the same individual. Thus we know that cold, and especially damp cold, is a cause of chilblains, but we do not know why *A* suffers from chilblains and *B* does not. Nor do we know why *B* did suffer from chilblains during the first World War but not since. We believe this to have been due to a scarcity of animal fats in the diet at that time but exactly how this lack caused chilblains we are ignorant. Similarly picric acid will cause a dermatitis when applied to the skin of *C* but not of *D* lacquer furniture will cause an eruption in *E* but not in *F* and so on.

The agencies or conditions causing skin diseases may be divided then into (1) predisposing, (2) immediate. Among pre-

disposing causes are diathesis, including psychological make-up, race heredity age sex, climate occupation and deficiencies in diet

The immediate causes of skin diseases are the following

*Congenital defects, or maldevelopment.*

*Physical causes*—pressure friction heat, cold radiant energy (sunlight, ultra violet light, X rays, radium)

*Chemical causes*—acids, alkalis and a host of other chemicals drugs, dyes tars, constituents of certain plants and woods, foreign proteins

*Animal parasites*—insects mites and worms.

*Vegetable parasites*—bacteria and coccid fungi, filter passing viruses.

*Protozoa.*

*Obstruction of vessels* (blood and lymph)

*Diseases of nervous system*—(a) central, (b) peripheral, (c) autonomic (d) psychical.

*Diseases of ductless glands.*

*Diseases of other viscera.*

Physical and chemical agents sufficiently powerful may produce eruptions on their first contact with the patient's skin, e.g. heat (burn) cold (frost bite) strong acids and alkalis. Very many other substances, of a less powerful nature may also cause dermatitis if allowed to come in contact with the skin in sufficient concentration for a sufficient length of time (primary irritant dermatitis). A further group may produce dermatitis by an allergic mechanism (sensitization dermatitis). Here the irritant substance which may be of quite a simple chemical nature acts as an antigen and is absorbed through the skin via the lymphatics, causing the formation of an antibody remote from the skin possibly in the lymph glands. The antibody is then transported back in the lymphocytes and transferred to the skin, there to react with fresh antigen the reaction resulting in dermatitis. These antibodies cannot be detected in the serum nor can they be transferred to a normal individual (negative Prausnitz-Kustner reaction). Finally some substances, after ingestion, inhalation or injection cause another type of cutaneous allergic

reaction known as urticaria. In this the antibodies can be detected in the serum and can be transferred to normal individuals (positive Prausnitz Kustner reaction)

There are a very large number of known antigens and probably an even larger number at present unknown, to which persons may become sensitised. Sensitisation is usually brought about by contact of the skin, especially if repeated with the antigen. The contact may be either external, or internal—that is, by the medium of the blood stream. Antigens may occur in foods (shell-fish) plants (*primula obconica*, "poison ivy" satinwood, teak), animals (e.g. cats, horses, animal parasites) and bacteria. To some substances the majority of people can be sensitised, e.g. salts of nickel 100% (Schittenhelm and Stockinger) sacaria 80% (Bloch) poison ivy 60% (Coca) to others the majority are resistant (pollens, *primula obconica*). However Bloch found that by concentrating an extract of *primula obconica* and painting this on a small part of the skin of normal persons he could sensitise the whole skin of every one of them. This suggests that whether a person becomes sensitive or not under given conditions is a question of (a) personal idiosyncrasy (b) dose of antigen, and that if the dose of antigen is only big enough no personal idiosyncrasy is required.

The reaction which the skin of a sensitised person shows to the antigen consists usually in dilatation of blood vessels (erythema) exudation of serum (oedema, urticaria) and vesication, if the exudation of serum is sufficiently great. The result of the reaction depends on the situation of the sensitised cells. If these are in the epidermis the result is eczema or dermatitis if in the dermis urticaria or erythema multiforme, if in the subcutis angio-neurotic oedema or erythema nodosum (Barber)

The skin eruptions usually accepted as being due to allergy are the following. Some forms of eczema and dermatitis, urticaria and angio-neurotic oedema, and drug rashes. Certain eruptions in persons with tuberculosis occurring at sites remote from the tuberculous focus and called tuberculides. Generalized eruptions occurring in patients with ringworm infections and called trichophytides and epidermophytides. Barber has attempted to

bring into line with the last two also the syphilides, and eruptions which he believes to be due to sensitisation to streptococci and calls the streptococoides

Lewis and his co-workers showed that injury of the skin always produced what he called the triple response. This consists in (1) a dilatation of the minute vessels in the area actually affected (2) an increased permeability of the vessel walls leading to exudation of serum and oedema (3) a spreading flush or "flare" on the surrounding skin brought about by dilatation of the "strong arterioles" by a local nervous reflex.

Lewis showed that the triple response is produced by the liberation from the injured cells of a substance similar to or identical with histamine which he calls *H* substance. This causes the dilatation and increased permeability of the minute vessels by direct action on their walls and the arterial flare through the intermedium of a local nervous mechanism.

The degree of injury necessary to produce a triple response is quite slight such as drawing a blunt point firmly several times along the same line on a normal skin or once only on the skin of a sensitive person, e.g. one who exhibits "dermographism". Injuries of other kinds, such as those due to freezing, burning, ultra violet light and caustic chemicals, produce a similar response.

In considering the pathology of diseases of the skin we have to consider the two principal layers of which the skin is composed, viz. the fibrous and vascular dermis and the cellular epidermis

The changes found in inflammatory conditions of the dermis are similar to those found elsewhere viz. dilatation of blood vessels diapedesis of leucocytes and proliferation of the fixed connective tissue cells. A prominent feature in sections from most skin diseases is the small round-celled infiltration, which is usually most marked around the blood vessels, the hair follicles and the sweat glands. These cells are derived partly from lymphocytes of the blood stream and partly from proliferation of the fixed connective tissue cells. Other cells which may be found in the corium in inflammatory conditions are polymorphonuclear

leucocytes and eosinophils derived from the blood stream and plasma cells endothelial cells, giant cells and mast cells (basophilic) derived from the tissues.

The changes which take place in the epidermis in inflammation are (1) An inter and intracellular oedema of the prickle-cell layer. This condition is known as spongiosis. (2) Owing to the tension put on the intercellular fibrils by the presence of abnormal amounts of fluid between the cells the fibrils may break down and the cells be forced apart. Also many of the epidermic cells liquify. In these ways minute vesicles are formed which increase in size until they become visible to the naked eye. They extend upwards until they lift up the impermeable horny layer. This is then easily rubbed off and leaves a moist weeping surface. Vesicles may also be formed by the swelling and rupture of the prickle-cells themselves from intracellular oedema. Vesicles and bullae may be situated at any level in the epidermis, either just above the papillae in the middle of the prickle-cell layer or just beneath the horny layer. (3) Even if the process does not proceed far enough to lift up the horny layer the oedema of the prickle-cells interferes with the normal process of keratinisation. A proper granular layer is not formed and the horn cells are moist sticky and retain their nuclei. Instead of falling off imperceptibly one at a time they stick together and form visible scales. The condition is known as parakeratosis and occurs to a greater or less degree in all forms of inflammation of the skin. In most types of parakeratosis, especially in psoriasis, leucocytes penetrate the epidermis from below and collect in masses between the layers of horn cells in the scales. (4) Normally cell division in the epidermis is limited to the basal layer but in certain forms of inflammation mitoses occur in the prickle-cell layer. As a result this is much increased in thickness, and its interpapillary portions push their way downwards between the papillae, whose height thereby appears increased. The interpapillary areas of the epidermis also become wrenched and irregular. This process is known as acanthosis. It occurs most strikingly in warts and molluscum contagiosum under the stimulus of a filter passing virus, and in chronic



inflammations. In all these conditions the prickle-cells are perfectly formed and provided with prickles, and the basal layer is intact.

There is, however another type of hypertrophy of the prickle-cell layer in which the cells are abnormal and have no interconnecting fibrils. The basal layer is imperfect and the multiplying cells penetrate it, reach the underlying corium and continue their proliferation there. This condition is consequently malignant and is, in fact epithelioma of the skin.

Injuries and diseases which affect the epidermis only and cause no loss of substance in the dermis heal without leaving any scar for they can be perfectly repaired by regeneration of the normal epidermic cells, e.g. warts impetigo. If however there is loss or destruction of the dermis it can only be repaired by granulation tissue. This turns into white fibrous connective tissue and a scar of some degree must result e.g. wounds ulcers.

Many chronic inflammations of the skin leave behind them when they heal more or less pigmentation. The pigment in such cases may be derived from haemoglobin, in which case it contains iron and is situated in the dermis. In other cases it is melanin and is formed in the basal layer of the epidermis, though it may pass downwards into the dermis.

The commonest organisms found upon the normal skin are shown in Table I on the opposite page. Normally they are all saprophytic but, under certain circumstances, the staphylococci may become pathogenic. The anaerobic bacteria outnumber the aerobic by 10-100 times and are found in areas of skin rich in sebaceous glands. *Staphylococcus aureus*, which is pathogenic is not normally present on the healthy skin, but is found in the anterior nares of 30-60% of healthy individuals. In boils and septic lesions, however this organism and haemolytic streptococci may be present in large numbers on the surface and in the mouths of the follicles of the skin not only around the lesion but also on other parts of the body. This fact explains why one boil is so apt to be followed by others and why persons with boils or impetigo even if only on face or neck should not undertake midwifery or the dressing of "clean" surgical cases.

TABLE I

## THE COMMON SKIN ORGANISMS (after Pillsbury and Kligman)

## 1 Aerobic flora

## A. Bacteria

*Staphylococcus epidermidis*  
*Staphylococcus albus*

## B. Lipophytic fungi

*Pityrosporon ovale*

## 2. Anaerobic Flora

*Propionibacterium acnes* (*Acne bacillus*)

The skin has a considerable power of self-disinfection mostly due as regards streptococcus pyogenes to unsaturated fatty acids in the sebum, as regards Gram negative organisms to desiccation, while both fatty acids and desiccation help in the destruction of staphylococcus aureus. (D. M. Pillsbury and A. M. Kligman.)

Psychological factors take a part in the causation of a number of skin diseases e.g. pruritus, eczema, urticaria, hyperhidrosis, vitiligo alopecia areata, lichen planus, psoriasis, acne excoriée rosacea, occupational dermatitis and dermatitis artefacta. Probably only few skin diseases are entirely produced by psychological causes but in particular patients psychological factors may be of great importance in perpetuating or aggravating the disease once started. They must therefore be taken into consideration in treatment.

## CHAPTER II

### SIGNS SYMPTOMS AND GENERAL DIAGNOSIS

The symptoms of skin diseases are itching and formication tingling, pricking, burning and pain. Hyperaesthesia par aesthesia and anaesthesia may also occur

Itching (pruritus) is the commonest symptom of skin disease. It may be continuous but more usually is intermittent and may be paroxysmal. The most characteristically itchy skin diseases are scabies pediculosis eczema, dermatitis, urticaria dermatitis herpetiformis, lichen planus, lichen urticatus, prurigo and mycosis fungoides. Itching may in some cases be the only complaint of the patient, no change in the skin being visible e.g. some cases of pruritus ani and vulvae. It may also be purely psychical in origin.

Formication is a variety of itching and is a sensation as though an insect were crawling about over or in, the skin.

Absence of itching is of assistance in diagnosis in some cases, e.g. syphilitic eruptions seldom itch while eruptions with which they may be confused, e.g. pityriasis rosea, psoriasis, may do so.

Tingling pricking and burning sensations are most commonly encountered in cases of eczema and dermatitis. Pain in herpes zoster and in inflammatory lesions such as boils and carbuncles. Hyperaesthesia, paraesthesia and anaesthesia may occur in lesions affecting nerves, e.g. herpes zoster nerve leprosy tabes dorsalis, syringomyelia. They may also occur in hysteria.

The physical signs of skin diseases are many and various. They are usually divided into primary and secondary lesions, but this division is largely artificial because a lesion for example a pustule, may be primary in one disease and secondary in another.

Atrophy of the skin may be primary or secondary generalised or localised. It usually results in the skin being more transparent and more shiny than normal and in wrinkling of the thinned horny layer.

Hypertrophy of the skin may be localised and involve principally the horny layer *e.g.* the callouses on the hands resulting from hard manual work or it may be widespread and involve all the layers, *e.g.* elephantiasis.

Swelling may involve mainly the corium, *e.g.* erysipelas and urticaria, or mainly the subcutaneous tissue, *e.g.* cellulitis and angioneurotic oedema.

Erythema (redness) is one of the commonest signs of skin disease. It is brought about by dilatation of both the strong arterioles and the minute vessels. The dilatation of the strong arterioles causes the blood flow through the minute vessels to be more rapid and the temperature of the skin is raised in consequence. Under certain degrees of cold, *e.g.* when handling snow the minute vessels may be dilated and the strong arterioles contracted. The rate of oxygen exchange between blood and tissues is at the same time reduced by the low temperature so that the blood remains arterial in tint. In such conditions a cold red skin is produced. Erythema may be punctate blotchy or generalised.

Cyanosis (blueness) is most common on the extremities. It is produced by a dilatation of the minute vessels in conjunction with a constriction of the strong arterioles. The circulation through the minute vessels is therefore very slow the blood in them becomes venous in character and the skin temperature is reduced, though not enough to keep the blood arterial in tint (*vide above*). Temperatures of about 15° to 25° C (59° to 75° F) usually lead to some degree of cyanosis in normal people, but a conspicuous cyanosis in response to cold is an abnormal reaction. (Lowia.)

Telangiectases are dilated superficial vessels, usually veins of the subpapillary plexus. They are common on the flush patches of the cheeks of normal people who have been much exposed to cold or to sun they are also a feature of old areas of X ray

dermatitis. Pressure with a watch glass forces the blood out of telangiectases and they disappear.

Echymoses are extravasations of blood from the minute vessels. They are most commonly found in purpura. Pressure does not alter their appearance.

Macules are areas of the skin which differ from their surroundings in colour or texture. They are not raised. Their colour may be redder or paler, browner or bluer than their surroundings, or their colour may be normal but the texture of their surface more wrinkled or more scaly. They may be of any size but are commonly  $\frac{1}{2}$  to  $\frac{1}{4}$  an inch in diameter.

Papules are essentially small raised lesions of the skin. They may be of normal skin colour or of any other colour. In plan they may be circular, oval or polygonal, and in elevation rounded, flat topped or pointed (acuminate). Their surface may be smooth, shiny, rough, scaly or warty. The summit of each may be crowned with a vesicle or pustule in which case they are described as papulo-vesicles or papulo-pustules. Papules are sometimes arranged in lines e.g. along a scratch in cases of lichen planus, plane warts or psoriasis.

Lichen is the name given to an aggregation of similar papules. These may be flat topped as in lichen planus, or pointed as in lichen spinulosus.

Lichenification is a condition of the skin produced by rubbing it over a long period. The colour is brown or violet, and the skin is thickened so that the lozenge-shaped areas between the normal creases of the skin stand up as flat topped papules somewhat resembling those of lichen planus. There may be some scaling. Lichenified areas irritate consequently the rubbing is continued and the vicious circle is complete.

Vegetations are masses of hypertrophic skin papillae or granulation tissue such that the appearance suggests the surface of a cauliflower. They are usually moist. When skin papillae are involved it is usually not the papillae themselves so much as the interpapillary areas of epidermis which are primarily hypertrophied; the papillae are drawn out passively. In conditions of lymphatic obstruction general (elephantiasis)

or local (warty lupus) probably lymphatic distension of the papillae is the primary condition. (Sampson Handley)

Nodules are small, usually rounded, lumps, in or on the skin e.g. those of lupus vulgaris, tertiary syphilis or leprosy

Tumours are larger lumps than nodules although there is no strict limit between the two.

Plaques are flat areas of the skin differing from their surroundings and usually harder than normal. They may be either raised above or depressed below the general surface, e.g. morphoea.

Wheals are areas in which the dermis is distended by oedema. They may be red or white or white with a red edge. The whiteness is the result of the collapse of the minute vessels under the pressure of the oedema. They vary in size from small papules to large plaques or they may be linear.

Vesicles are small blisters filled with serous fluid. They may be so small as to be barely visible to the naked eye or may be several millimetres in diameter. They may be either tense or flaccid and their roofs may consist of horny layer only of the whole epidermis, or of any intermediate amount of the Malpighian layer in addition to the horny layer. A vesicle is often set on the summit of a papule.

Bullae are large vesicles. The term is generally used only of vesicles more than about 1 cm. in diameter. They may be many inches across and on rupture leave large raw areas of skin. They may be tense or flaccid and their contents may become purulent.

Pustules are small blisters filled with pus or small collections of pus not actually on the surface. They may be situated on the summits of papules forming papulo-pustules, at the mouths of the pilo-sebaceous follicles (sycomas) about the upper third of the follicles (acne vulgaris) or around the bottom of the hair follicles (boils).

Abscesses are collections of pus too large to be called pustules, i.e. more than a few millimetres in diameter.

Cysts are non-inflammatory collections of material, which may be fluid or semi-solid, surrounded by a well-defined wall. They may vary in size from a millimetre or so up to several

inches across. Their contents most commonly consist of horn cells (milia) or sebaceous material (sebaceous cyst)

A comedo or blackhead is an oat shaped structure occupying the mouth of a pilo-sebaceous follicle. It is made up of concentric layers of horn cells and sebum and contains many acne bacilli. Its outer end is usually dark-coloured from deposition of pigment, and its inner end on expression may tail off into a worm like structure composed of soft sebum. Many comedones contain a microscopic mite, *demodex folliculorum* which is harmless. Comedones are the characteristic primary lesion of acne vulgaris. They also occur in work people exposed constantly to chlorine or chlorinated naphthalenes, tar or heavy mineral oils. Grouped comedones occur in infants as a result of rubbing with camphorated oil or tallow or of contact with dirty and greasy clothing.

The burrow is a lesion characteristic of scabies. It appears as a wavy line generally about 1 cm. in length on the surface of the skin. In colour it is usually whitish with darker specks here and there. At one end a minute white shiny speck with a dark nose may be seen which is the acarus. Parts of the burrow are often incomplete, the roof having been removed by washing. Burrows are most commonly found on the front of the wrist and ulnar border of the hand (Scabies, p. 132).

Keratoses are thickenings of the horny layer of the skin. They may appear as small yellowish or dark-coloured warty excrescences on the face or backs of the hands in those who have worked for many years exposed to sunlight, tar, arsenic or crude mineral oils. Or they may consist of a hypertrophy of the horny layer on the palms and soles around each sweat opening, e.g. in those who have taken arsenic internally for years.

Weeping is a condition which arises when the horny layer has been removed from the skin and lymph or serum exudes from between the exposed prickle-cells. The horny layer may have been removed by injury (abrasion) or have got rubbed off after having been loosened by the accumulation of serum beneath it, either as a multitude of minute vesicles (eczema) or as large single vesicles (impetigo).

Crusting takes place when the serum from a weeping surface dries up. The crusts often contain some horn cells or some pus cells.

Scaling (parakeratosis) (p 5) is a common sequel to inflammations of the skin. The scales consist of masses of horn cells often with some leucocytes between them. They differ therefore from crusts which are essentially dried serum. The two however are often combined as scale-crusts.

Pigmentation of the skin may be primary or secondary to a previous inflammation. It may be due to melanin (p 6) or to pigment derived from the blood, or to foreign bodies (silver in argyria, carbon or cinnabar in tattooing)

Depigmentation with the formation of white patches may occur in vitiligo and in syphilitic leucoderma of the neck. In such conditions there is often an excess of pigment in the area immediately surrounding the white patch.

Circinate lesions are those in the form of circles, or arcs of the same. The circle may be erythematous, pigmented or scaly or it may be made up of papules, nodules, vesicles, or pustules.

Fissures are cracks in the epidermis usually involving the dermis to some extent also. They commonly occur at the angles of the mouth and nose where they are usually associated with streptococcal infection. Also behind the ears, in the natal cleft, and on the palms and soles.

Ulcers are areas where there is a loss not only of the epidermis but also of some of the dermis. They are commonly the result of infection of a normal skin (e.g. ecthyma, tuberculosis, syphilis) or of malnutrition of the skin (e.g. varicose ulcer).

Scars are left when the dermis has been damaged by injury or infection. Lesions which have affected the epidermis only do not leave scars. Scars may be thin and depressed below the general surface (e.g. those of tertiary syphilis) or thick and raised above it (keloid). When recent they are red when old they may be white or pigmented. Several diseases leave characteristic scars, e.g. lupus erythematosus (p. 305) syphilis (p. 214) herpes zoster (p. 226).

Scratch marks are commonly seen in all itching diseases. They are linear excoriations which may become infected. They



are perhaps most strikingly seen on the shoulders in cases of pediculosa corporis. In warts lichen planus, and psoriasis, the line of a previous scratch is often indicated by the presence of a line of the papules characteristic of the disease.

### GENERAL DIAGNOSIS

It is a good practice to ask the patient first of what he or she complains and to take a preliminary look at the eruption. The object of this is to ascertain the type of condition one has to deal with. If one starts by taking a history without a preliminary examination one may fail to elicit material points as the result of getting a totally wrong idea of what the patient is talking about, e.g. patients almost invariably describe wheals as "blisters." Moreover it prevents waste of time in taking a long history in conditions of obvious etiology such as scabies or pediculosa. Having taken a preliminary look at the eruption one then settles down to get a detailed history. This should cover the following points (1) age (2) married or single (3) occupation. Try and find out exactly what the patient does. "Packer" for example is insufficient. One needs to know what he packs and what he packs it in—hay straw paper etc. O'Donovan's tea boy with dermatitis of the ankles and feet is a characteristic example. On enquiry it appeared that the boy's duty besides making tea was to carry it to the men engaged on a new building. His socks got filled with cement dust and his trouble was a cement dermatitis. (4) Duration of present occupation (5) Previous occupations. The importance of this is that some skin conditions (e.g. chronic solar dermatitis tar carcinoma and mule spinner's cancer) have a latent period of many years. (6) Duration of disease. (7) Mode of onset site subsequent spread etc. (8) Does it itch, if so when? (9) Whether eruption is continuous or intermittent. (10) Has patient undergone any special nervous shock or period of stress? (11) To what patient ascribes the origin of the disease? (12) In chronic diseases what makes it worse and what makes it better? (13) Has he had it before. (14) Are any others in family in house or doing the same work, affected.

(15) Digestion, bowels menstruation, sleep etc. (16) Previous illnesses. (17) Has patient ever lived abroad especially in the tropics. (18) Family history of similar or allied conditions. (19) Habits, alcohol, tobacco exercise fondness for sweets, tea, etc. (20) Hobbies, gardening, etc. This is important, because the patient's trouble may arise not from his work but from his play. (21) Whether patient is receiving, or hopes to receive compensation for his illness.

Having got these details one knows something about the patient, and while eliciting them one has been able to get an idea as to whether he or she is intelligent, observant, trustworthy neurotic, hypochondriacal, optimistic or the reverse.

It will obviously not be necessary to go into the history in such detail in all cases but even where the diagnosis is obvious it is a good thing to take a fairly complete history for the sake of what one can learn from it about the patient. For after all it is the patient one has to treat, not the disease.

One then proceeds to the physical examination of the patient. For this a good light is absolutely essential. Ideally one should see the whole surface of the patient, but this is obviously unpracticable in most cases and generally not really necessary. In cases where the diagnosis is in doubt, however one should insist on seeing as much of the patient's body as one thinks necessary. Patients statements that there is "nothing any where else" are not necessarily true. They may be dictated by modesty laziness, consciousness of dirty underclothes, or mere lack of observation. It is remarkable how some patients will come many miles to consult a doctor and then be disinclined to be properly examined.

One should note first of all the distribution of the eruption. Many diseases have a characteristic distribution, others are not so particular. Then one should note, in the following order the characters of the eruption. By inspection, its configuration, whether diffuse or in discs, circles, etc. the type of the individual lesion, whether macules, papules, vesicles, etc. their colour, shape, edge whether sharp or indefinite, and the nature of their surface, whether shiny dull or rough. By palpation one should

then determine whether they are situated on in, or under the skin whether they are hard, soft, elastic fluctuating, etc.

Experience has shown me that students, and (low be it spoken) even practitioners, quite commonly fail to observe accurately the nature of the individual lesions of which an eruption is composed and so fall into grievous errors of diagnosis. Such errors are the diagnosis of herpes simplex as impetigo of circinate impetigo as ringworm or lichen planus as psoriasis, to mention a few of the commonest. One cannot lay too much stress on the necessity for accurate systematic and detailed observation in dermatology.

A hand lens is of great use in detecting the exact character of an eruption, and if the room is not very well lighted an electric torch is a help also.

Having ascertained the nature and distribution of the eruption and the absence or the presence and character of any enlarged lymph nodes one should then mentally tabulate the possible diseases which would fit in with these for which purpose the diagnostic index at the beginning of this book may be of some assistance. One has to bear in mind the probabilities due to the patient's diathesis (as indicated by appearance, manner and history of other diseases) race heredity (as revealed by family history) age sex, occupation and the climate in which the patient lives or has lived. Having decided upon two or three possible diagnoses one should then search the patient afresh for supporting or rebutting evidence. As an example if there is a doubt whether a patient has psoriasis or lichen planus the discovery of white spots and streaks on the buccal mucosa would support lichen planus, while the discovery of small pits on the nails like the markings on a thumb would be in favour of psoriasis. Similarly if it is a question whether a scaly scalp is due to seborrhoea or psoriasis the discovery of pits on the nails would support the latter as against the former. The late Dr John Biernacki, who taught "fevers" to me and many of my contemporaries, used to lay stress on the value of taking a piece of paper and jotting down in parallel columns the points in favour of and

against each of two possible diagnoses. He said, and I have found it true, that if one does this the evidence is found in the majority of cases to point strongly to one alternative as against the other. Incidentally this is an excellent method of deciding between two courses of action in life generally.

Having arrived at a provisional diagnosis one then completes the general examination of the patient as regards pulse, tongue, teeth, tonsils, nose for obstruction, scalp for scurf, reflexes and urine. One should take the temperature and examine the circulatory and nervous systems, the chest and abdomen, in detail if there appears to be any need for so doing. Ideally one should make these examinations in every case but as there are only twenty four hours in the day one usually omits them in the absence of special indications. One has always to bear in mind, however, that the best way to miss something is not to look for it and the more complete and conscientious one's examination the greater confidence one can have in one's diagnosis.

Special examinations which may be indicated in particular cases are the following: *Diascopy* i.e. examination of a lesion while under pressure with a piece of glass, e.g. watch glass. This presses the blood out of the vessels and is useful for detecting the apple-jelly nodules of lupus vulgaris, to take one example. *Wood's light*. This is the light from a mercury vapour lamp or other source of ultra violet light passed through a piece of Wood's glass. The latter is a dark violet, nearly black, glass containing oxide of nickel. It transmits no light except the long ultra violet rays of wave length from about 3600 to 3300 Angstrom units, with in addition some visible violet and a little red. The ultra violet component in this light causes hairs infected with small-spored ringworm to fluoresce brilliantly with a greenish light if examined in a dark room (pp. 41, 174).

Microscopic examination of hairs or pieces of the horny layer of the skin warmed in liquor potassae is necessary to confirm a diagnosis of ringworm or of other fungus infections (p. 171). Microscopic examination is also necessary to confirm a diagnosis of scabies (p. 132).

then determine whether they are situated on, in, or under the skin whether they are hard, soft, elastic fluctuating, etc.

Experience has shown me that students, and (low be it spoken) even practitioners, quite commonly fail to observe accurately the nature of the individual lesions of which an eruption is composed and so fall into grievous errors of diagnosis. Such errors are the diagnosis of herpes simplex as impetigo of circinate impetigo as ringworm, or lichen planus as psoriasis, to mention a few of the commonest. One cannot lay too much stress on the necessity for accurate, systematic and detailed observation in dermatology.

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## CHAPTER III

### GENERAL TREATMENT

THE treatment of skin diseases must be considered under the following heads: general management; food and drink; elimination of septic foci; drugs by mouth; drugs by injection; external applications; physical methods.

**General management.** Acute or extensive skin diseases such as eczema, dermatitis, lichen planus, psoriasis are best treated with the patient in bed. This secures rest for the body. Rest for the mind is in many cases equally important but not so easy to obtain. Removal from home or business worries or from uncongenial or too sympathetic persons, is often best secured in a nursing home or hospital. Rest in bed also reduces friction from clothing, and allows of more effective application of external remedies.

The bowels should in general be kept well open, saline aperients being useful for this purpose.

If the patient is not sent to bed the clothing should be loose and not too hot.

The best climate for the patient will depend upon the nature of his disease. For example, sufferers from superficial sepsis or from tuberculous require plenty of sunlight, those with lupus erythematosus and eruptions produced by light should avoid a sunny climate.

Hard water is irritating to many skins and should be avoided if possible, but the addition of most "water-softeners" or of "bath salts" (sodium carbonate) to the water is not desirable. Frequent washing with plenty of soap and hot water is required in acne but must be carefully avoided in eczema and dermatitis.

The patient's usual occupation or recreation may be the cause

of or may aggravate his trouble and may need to be temporarily suspended or given up altogether

**Food and drink** A properly balanced diet is necessary for health, but given this, I am not convinced that food and drink have in general the great effects on skin diseases which are often ascribed to them. There are however particular conditions on which diet has a definite influence. Acute urticaria for example, is often due to some food shell fish being the most usual offender. There are however few foods which have not at some time produced urticaria in somebody.

Some patients with eczema notice that they cannot eat certain foods for example rhubarb tomatoes and sour fruits, or drink certain wines without bringing on or aggravating an attack. Many middle-aged patients with (and without) eczema eat and drink too much and an all round reduction of food and alcohol proves beneficial. Alcohol tends to aggravate most skin diseases, partly because of its dilating effect upon skin vessels and partly perhaps because it promotes absorption from the alimentary tract of protein fractions which should not normally be absorbed. The deleterious substance in many wines and beers however seems not to be the alcohol itself for many patients who cannot take heavy wines or beers without aggravating their eczema can take whisky and water with impunity. Eczema is aggravated by pungent substances such as ginger but I am doubtful whether the usual small amounts of pepper and mustard make any appreciable difference. Over ripe cheese and high game should also be avoided. A low salt diet is sometimes of use in eczema and oedema, and good results have also been claimed for it in lupus vulgaris (p 205) but these claims do not seem very well founded.

Sufferers from acne vulgaris and seborrhoea are often improved by cutting down their carbohydrate intake. Girls who are honest with themselves often notice that their "spots" are worse after they have consumed a boxful of chocolates. Whitfield held that this is due to the cacao butter of which ordinary chocolate contains some 40% by weight. Certainly "boiled sweets" do not seem to have the same bad effect. A

few patients with acne vulgaris are made worse by eating cheese, but this in my experience is uncommon.

Patients with rosacea are sometimes found to consume excessive quantities of strong tea, or less commonly nowadays of alcohol and the substitution of other drinks is an essential part of their treatment.

Elimination of septic foci, Barber especially has called attention to the importance of septic foci in originating or perpetuating some skin diseases such as chronic urticaria, lupus erythematosus, and erythema multiforme. The most common sites of such infections are the roots of the teeth the tonsils and the nasal sinuses, but other places such as the appendix the urogenital tract, and even, as in one of Barber's cases, the scar left by an old carbuncle, must not be forgotten. I have seen two cases of rosacea clear up suddenly after the removal of infected teeth. The infecting organism is usually a streptococcus.

Drugs by the mouth have a certain limited value in dermatology. Dapsone (diamino-diphenyl-sulphone) is the treatment of choice in dermatitis herpetiformis. It is also used in the treatment of leprosy. Arsenic, given as liquor arsenicalis, is rarely if ever used in the treatment of dermatitis herpetiformis on account of its effects if given over a prolonged period of time, but it still has a place in the treatment of pemphigoid. Chloroquine and sometimes mepacrine, are used in the treatment of lupus erythematosus and in eruptions caused by light. Isoniazid (isonicotinic acid hydrazide) is the most satisfactory treatment for lupus vulgaris and other types of tuberculosis of the skin.

The sulphonamides are little used in dermatology nowadays, their place having been taken by the antibiotics. Sulphapyridine, however, has a specific effect, pharmacological as opposed to anti bacterial in dermatitis herpetiformis and is sometimes used in the treatment of this malady.

Calcium has a reputation in the cure of urticaria, chilblains and plane warts. I have seen several cases where plane warts disappeared after the patient had taken half a pint of lime water



daily for some weeks, and injections of calcium gluconate sometimes appear to reduce irritation in acute eczema.

Iodine is of value given in large doses in fungus infections such as actinomycosis, blastomycosis, and sporotrichosis. It may be given as Pot. iod. or as a 10% solution of iodine in spirit. In dermatitis herpetiformis and pemphigus it is contra-indicated. Mercury by the mouth is useful in lichen planus and occasional in plane warts as well as in syphilis.

Yeast and its extracts are often given in cases of boils and more usefully in cases requiring the vitamin B complex.

Sedatives and hypnotics are often required in dermatology to allay itching and procure sleep. Aspirin, chloral, phenobarbitone, medinal, sodium amytal, soneryl, other barbiturates and the bromides all have their place. Opium, although not used to allay itching is of value in some cases of pemphigus. Certain drugs, known as tranquillizers which are advocated for the relief of agitation and anxiety but which are neither hypnotic nor sedative, are also sometimes useful. Examples are chlorpromazine hydrochloride ( Largactil ) Meprobarbital (" Equanil " Miltown ) and methyl pentynol ( Oblivon ). On the whole however small doses of barbiturates such as amytal gr  $\frac{3}{4}$  or phenobarbitone gr  $\frac{1}{2}$  two or three times a day are more effective.

Of the large number of antihistamines, the most useful is meprobamine maleate (" Anthusan ") which has a mild hypnotic action and is effective for about 4 hours. chlorcyclizine hydrochloride ( Histantin ) which has almost no hypnotic action and is effective for 8 hours and promethazine hydrochloride ( Phenergan ) which is effective for 12 hours, but has a marked hypnotic action and should only be given at night. They are useful mainly in urticaria, but also have a slight antipruritic effect. There are also large numbers of other antihistamines available and certain patients are found who obtain benefit from one and not from another.

Of the gland products thyroid extract and adrenalin are the two most useful. Thyroid extract is given with good effect to patients with loss of hair due to hypothyroidism also to

patients with scleroderma, occasionally with benefit. It is largely used also in chilblains and in all sorts of other diseases without much justification. Adrenalin and its vegetable substitute ephedrine have a good effect on some cases of urticaria even if taken by the mouth. Natural and synthetic oestrogens are sometimes useful in pruritus vulvae and in other conditions in women past the menopause, also in some cases of acne vulgaris in young people. Methyl testosterone is useful in generalized pruritus, especially when due to jaundice.

Injections of one sort or another are used a good deal in dermatology. Vaccines were and are largely used by some. So far as my own experience goes I have seen occasional good results in furunculosis and pustular acne.

The appropriate sera are of great value in cutaneous diphtheria and anthrax.

"Protein shock" is used in some chronic skin diseases and is often followed by temporary improvement. It is also used in attempts to desensitize patients with chronic urticaria or eczema. Slight degrees of shock may be produced by injections of 5-10 c.c. of the patient's own blood intramuscularly into the upper part of the buttock every 5-7 days, or by 5-10 c.c. of well boiled milk in the same situation. The best site for injection is in the middle third of a line joining the anterior superior spine of the ilium with the top of the natal cleft. Greater degrees of shock are produced by intravenous injections of typhoid vaccine (T.A.B.) in doses of 50-800 million organisms or more once or twice a week. The first dose should be 50 million organisms and later doses should be increased as necessary to give a rise of temperature to about 102° F (39° C) each time. If less shock is desired the vaccine can be given subcutaneously or in smaller doses.

Bismuth is still used by some in the treatment of syphilis. It is given intramuscularly in this country usually as a suspension of the metal in glucose solution. It is used with good effect also in lupus erythematosus.

Mercury is now seldom used by injection in syphilis. It is useful in lichen planus.

Arsenic is still used in the form of the arsenobenzenes in syphilis. It can also be used effectively in lichen planus in the form of mercury salicyl arsenate ("Enecol"). In dermatitis herpetiformis I have not found injections so reliable as Fowler's solution by the mouth.

Antimony as tartar emetic (potassium antimonyl tartrate) or the equivalent sodium salt intravenously is useful in cutaneous leishmaniasis and in granuloma inguinale.

Gold is given intravenously as the chloride as gold-sodium thiosulphate (Sanocrysin) or in organic combinations such as Lopion "Solganal, etc. Nowadays, however it is usually given intramuscularly as gold sodium thiomaleate (Myocrisin), its principal use being in the treatment of lupus erythematosus.

BAL (British Anti Lewisite) 2-3 Dimercaptopropanol, by intramuscular injection is very useful in cases of metallic poisoning due to arsenic, gold mercury or bismuth.

Drugs applied externally have a large part in the treatment of skin diseases. Those most used are the following

**Antipruritics** Carbolic acid 1-20 to 1-80 liquor picis carbonis tars, menthol chloroform crotonol ( "Furax" )

Quotane, hydrocortisone, menthol, thymol and camphor

**Antibiotics** are sometimes used successfully by external application as ointments e.g. the tetracyclines, polymyxin, chloramphenicol and neomycin.

**Antiseptics** Boric salicylic and benzoic acids, the salts of mercury hydrogen peroxide eusol iodine sulphur resorcin potassium permanganate copper sulphate, silver nitrate and dyes such as flavine brilliant green crystal violet and fuchsin. Carbolic acid one seems to use more often as an antipruritic or as a caustic than as an antiseptic

**Astringents** In solution, lead subacetate zinc sulphate copper sulphate alum silver nitrate In powder form, zinc oxide, zinc carbonate (calamine) bismuth subnitrate or oxycarbonate

- Caustics** *Strong* Nitric acid trichloroacetic acid, acid nitrate of mercury zinc chloride, pure carbolic acid.
- Weak.* Glacial acetic, chromic, salicylic and pyrogallio acids, liq potassae, silver nitrate potassium permanganate copper sulphate, resorcin.
- Keratolytics.** For removal of horny thickening Salicylic acid, liq potassae soft soap water
- For removal of hair Barium and strontium sulphides Calcium thioglycollate
- Lubricants** Lard lanoline petroleum jelly liquid paraffin, olive and sweet almond oils cold cream.
- Parasitocides** *Animal.* Benzyl benzoate, D.D.T., sulphur balsam of Peru, xylol, alcohol carbolic acid, mercury  $\beta$  naphthol naphthalene, "Lethane"
- Vegetable.* Salicylic and benzoic acids, chrysarobin, dithranol silver nitrate iodine fuchsin, brilliant green, crystal violet.
- Protectives** Powders as such or incorporated in pastes and lotions: zinc oxide, zinc carbonate (calamine) bismuth oxycarbonate or subnitrate talc.
- Paints.** Collodion, zinc gelatine.
- Stimulants** Tar, e.g. prepared coal tar Ol. cadini (juniper tar) Pix liquida (pine tar Stockholm tar) Ol. betulae or Ol. rusci (birch tar) Ol. fagi (beech tar) Liq. picis carbonis.

The types of external applications used in dermatology are as follows

**Baths** Continuous baths in which the patient remains immersed up to his neck for weeks at a time are much used in Vienna, but not in this country. They are used chiefly in cases of extensive dermatitis and of pemphigus.

Saline baths are useful in cases of extensive scapula or of dermatitis. They should be made slightly hypertonic by adding three pounds (1.5 kg) of common salt to a thirty gallon (135 litre) bath and should be of a comfortable warmth, say 100° F

An antiseptic bath, useful in cases of boils, may be made by dissolving 4 ounces (116 grm) of zinc sulphate in a 30 gallon bath.

The best way is to dissolve the zincsulphate first in a jug of boiling water and then add this to the bath (Whitfield.)

Antipruritic baths may be made with bran or starch. Two to four pounds (1.2 kg) of bran for a thirty gallon bath should be put in a very loose muslin bag and tied under the hot tap the water being run into the bath through the bag which should be squeezed at intervals. If put loose into the bath the bran is apt to clog the waste pipe.

A starch bath is made by making two pounds (1 kg) of starch into a paste with cold water and then running the hot water into the bath through the basin of starch.

Alkaline baths are useful for removing crusts. Four ounces (116 gm.) of sodium bicarbonate should be added to thirty gallons (135 litres) of water. Tar baths, made by adding two to four ounces of solution of tar are much used in psoriasis.

Some patients with extensive pruritus find that very hot baths, as hot as can be borne are soothing and if taken at bed time allow them to get to sleep.

Soaps have a limited application in dermatology. Ordinary toilet soaps, soft soap (*sapo viridis*) or ether soap are useful in *acne vulgaris* to remove excess of sebum and of horny layer. Soft soap is useful in psoriasis to remove scales and in scabies to remove the horny layer forming the roofs of the burrows.

Medicated soaps are principally of value in that a patient ordered a medicated soap is more likely to use it thoroughly than if he is told to use any good toilet soap. The medicament incorporated in the soap by the time it is diluted with water to form a lather is usually in such low concentration as to have very little effect. This applies particularly to "antiseptic" soaps. An exception must be made in favour of Tetmosol soap in the prophylaxis of scabies (v p 140).

Spirit soap (*saponis viridis* two parts, industrial spirit one part) is useful as a shampoo in seborrhoea and psoriasis of the scalp. Soapless shampoos (sulphonated lauryl, etc) are useful especially with hard water.

Superfatted soaps containing lanoline are preferable to

ordinary toilet soap in patients who have dry skins or are inclined to eczema or to "chapping" of the hands.

Powders are used to dry the skin, to cool it (by providing increased radiating surface) and to prevent friction between adjacent surfaces. Talc powder zinc oxide, starch and bismuth subgallate are all commonly used.

Lotions are largely used in dermatology. They may be cooling, astringent, antiseptic or antipruritic. Examples are

Loto plumbi, viz. R.	Lq plumbi subacetatis fort.	3 i.	1
	Aqua dest. ad	1 pint.	160
Eau d Albour viz. R.	Zinc sulphate	gr vi.	12
	Copper sulphate	gr iv	1
	Camphor water to	3 L	100

Also lotio rubra and carboleo lotion 1 20 to 1-80

The widely used varieties of calamine lotion combine the properties of a lotion and of a powder e.g.

R.	Calaminae prep	3 i	12 parts.
	Zinci oxidl	3 i	12 parts.
	Lq plumbi subacet. dil.	3 L	12 parts.
	Glycerini	3 L	12 parts.
	Lq calois ad	3 L	100 parts.

Bentonite (B.P.O.) 2-5% may be added as a suspending agent. The evaporation of the water cools the skin, the lead is soothing and astringent, the calamine and zinc oxide remain as a protective covering and the glycerine makes them adhere.

In general lotions should be dabbed on and allowed to evaporate and then be renewed. They may however be applied as wet dressings.

Wet dressings for raw surfaces may be made with lead lotion lotio rubra, eusol, or flavine according to the effect desired. Tulle gras interposed between the raw surface and the wet dressing will prevent them from sticking together. If a cooling effect is desired no waterproof covering should be used and only as little bandage as possible.

For septic lesions wet dressings of 12% urea or sodium sulphate are particularly useful.

Boric fomentations are useful in septic conditions. They should be renewed every quarter of an hour for say an hour at a time two or three times a day. The skin should be wiped over with an antiseptic lotion or with methylated spirit after each application otherwise fomentations are apt to spread such conditions as boils or impetigo. In the interval between periods of fomentation the area may be covered with dry cotton wool heated before application. Fomentations of 14000 hydrarg. perchlor are also useful.

Kaolin poultices are often preferable to fomentations.

Starch and borio poultices are valuable for removing crusts. They are made as follows. Mix two teaspoonful of boric acid powder and four tablespoonful of wheat starch (*Pulv amyli*) with cold water into a thin paste in a bowl or cup. Take another bowl and ascertain to what level up its side a pint of water reaches mark this with adhesive strapping and empty the bowl again. Then pour into it simultaneously the thin starch paste and water from a boiling kettle until the level representing one pint is reached. Stir vigorously until the starch grains burst shown by a sudden increase in the translucency of the solution. Then set aside until cold. The resulting jelly should be turned out, cut into slices half an inch thick and applied between layers of gauze or muslin. The application should be renewed four hourly.

Creams and liniments are very useful preparations in inflamed states of the skin. They should be smeared on thickly and lightly covered or applied on gauze or butter muslin. They have to some extent the cooling effect of calamine lotion but owing to their oily nature do not dry up so much.

#### *Zinc Cream*

Zinci oxid	3ii	25
Adipis lanæ hydros.	3ii	25
Ol. olivæ } Liq calcis }	a. a. ad 3i	100

#### *Calamine Liniment*

Calaminæ	3ii	25
Adipis lanæ anhyd	gr v	1
Acid oleo	m. nls	0.5
Ol. olivæ } Liq calcis }	a. a. ad 3i	100

Oils are useful to soften crusts and in conditions of excessive dryness of the skin. Olive oil sweet almond oil and liquid

paraffin are those most used. Ten per cent. of oil of eucalyptus in lard is excellent for softening crusts in impetigo.

Ointments are preparations consisting mainly of a greasy base but containing varying amounts of solid or liquid active ingredients. The greasy base causes the ointment to adhere and enables it to be spread evenly over the required area. The bases most used are benzoinated lard, hydrous lanoline, yellow or white soft paraffin, mixtures of hard and soft paraffin melting at any desired temperature, mixtures of lanoline and petroleum jelly and emulsifying bases.

Lard and lanoline are absorbed by the skin, although the latter should be thinned with lard or olive oil if absorption is desired. The mineral hydrocarbons, such as the paraffins, are not absorbed and therefore keep the skin greasy.

Lard being an animal fat and saponifiable by alkalis is more easily removed by soap and water than are the paraffins. This may be an advantage or not according to circumstances.

Ointment bases may be divided into those which are not absorbed by the skin such as soft and hard paraffin, beeswax and mixtures of these, those which are absorbed such as benzoinated lard, lanolin thinned with olive or arachis oil, coconut oil and the emulsifying bases. The latter are of two main types: those which produce water in-oil and oil in water emulsions respectively. Wool alcohols are the unsaponifiable fraction of wool fat. 6% of these mixed with hard soft and liquid paraffin constitute the B.P. Ointment of Wool Alcohols (Eucerin anhydrous) and this with equal parts of distilled water becomes Hydrous Ointment B.P., a water in-oil emulsion (Eucerin hydrous). Emulsifying Wax B.P. is 10% sodium lauryl sulphate and 90% ceto-stearyl alcohol (Lanette wax S.X.). Emulsifying Ointment B.P. consists of 30% emulsifying wax with soft and liquid paraffin (Halden's Emulsifying Base H.E.B. simplex). Hydrous Emulsifying Ointment B.P. consists of emulsifying ointment 30% water 69.9% chlorocresol 0.1% (to inhibit the growth of moulds). This is an oil in-water emulsion. The approximate commercial equivalents of the



above preparations are shown in brackets. A useful base for penetrating ointments is a polymer of ethylene glycol resembling white paraffin known as Carbowax 1500 (Carbide & Carbon Chemicals U.S.A.) It is water-soluble and itself dissolves many antiseptics and is therefore very useful for the treatment of diseases of the scalp. The addition of a wetting agent e.g. 10% of Crill No. 6 (Croda Ltd. Wool Fat Products 449 Grand Buildings London, W.C. 2) which is miscible both with water and vegetable and mineral oils increases the penetrative power of Carbowax 1500 and is useful in the treatment of ringworm of the scalp (p. 176). Carbowax is sometimes irritating to diseased skins.

When ointments are required to protect the skin they should be made with the non-absorbable paraffin type of base. When they are required to replace the natural sebum they should contain some ointment of wool alcohols or else lanoline thinned with olive arachis or coconut oil. When required to carry some medicament into the skin they should be made with benzoinated lard or with the water in-oil type of emulsifying base or preferably with Carbowax 1500 with or without 10% of Crill No. 6. When required to carry an antiseptic for a moist oozing surface the oil in water type of emulsion base is best. This type is also best for use on the scalp as it washes out more easily than the water in-oil type. The concentration of a medicament used in either type of emulsifying base should be less than if used in a paraffin base on account of the improved absorption. Some skins are irritated by too great a concentration of emulsifying wax in an application. Various substances such as phenol (more than 1 or 2%) menthol ichthammol and liquor picis carbonis tend to break down the emulsion in hydrous ointment.

Ointments tend to heat the skin as they obstruct the radiation of heat and the evaporation of water. They also soften the horny layer.

Glycerine of starch can be used as a non greasy base for ointments. It is easily removed by water.

Cold creams (Ung. aquosum B.P.) are ointments containing

about 50% of water. The evaporation of this cools the skin. They are useful also for cleansing the skin in conditions when it is easily irritated by soap and water.

Pastes are preparations in which the solid ingredients and the greasy base are present in about equal parts. They are less heating to the skin than ointments, do not cause the same degree of maceration of the epidermis and form a more protective covering. Owing to their content of powder they are supposed to be able to absorb exuding fluid to a certain extent. The most commonly used paste is that devised by Lassar viz

R.	Zinci oxidi	3 fl.	25
	Pulv. amyli	3 fl.	25
	Adipos. lanae hydros.	3 fl.	25
	Petroleum jelly	3 l.	100

As some skins are irritated by lanoline this may be replaced by Petroleum jelly as in the B.P. Zinc Oxide Paste.

Originally this paste contained *Acid. salicyl.* gr. x, but this is usually omitted. Various drugs such as tar sulphur and resorcin may be added to this preparation. Pastes should be thickly applied and covered with as little bandage as possible. In Vienna no bandage is allowed, and the surface of the paste is merely dusted with powder.

Paints have a limited field of use. They are cleanly but drugs incorporated in them are less effective than in ointments. The bases used are collodion and traumaticin (gutta percha 3 i chloroform 3 i, s.s. 1.8). Unna's zinc gelatine (gelatine 15 glycerine 10 zinc oxide 25 water 50 parts) and Pick's varnish (tragacanth 8 glycerine 2 water 92 parts) are useful preparations in which various medicaments may be incorporated.

Plasters containing salicylic acid (10%-40%) are sometimes used for reducing horny thickening and for plantar warts and those containing mercury and carbolic acid for boils.

Three factors must be considered in deciding what application to use in any given case (1) The drug to be used (2) the strength in which it should be applied, (3) the type of vehicle most suitable s.e. lotion, ointment, paste, etc.

1 In deciding upon the drug to be used it is important to remember that in many common conditions caused primarily by infection, *e.g.* ringworm of the feet and impetigo the skin after a time becomes "eczematized" and very sensitive to substances which would not have irritated it originally. In such cases if one starts treatment with antiseptics one may aggravate the eczematous element in the condition and make the patient worse. It is better to start with soothing remedies such as zinc cream until the eczematous element has been subdued, and then gradually add the desired antiseptic.

2 Patients' skins vary enormously in their sensitiveness to drugs and it is wise therefore always to start with a weak concentration and increase it as necessary. If one uses too great a strength to begin with and it proves irritating, one is left in doubt as to whether the drug or its concentration is at fault. This is a matter in which experience is the best teacher.

3 The type of preparation to be used should be decided in general on the following lines:

Erythema, wheals, oedema, and vesicles require powders or lotions.

Weeping surfaces require lotions, liniments or creams.

Crusts require oils or ointments or else fomentations or starch poultices.

Septic lesions require baths, fomentations, wet dressings, or starch poultices.

Scales require ointments.

Papules require lotions, ointments or pastes.

Subacute stages of eczema and dermatitis require pastes.

Pruritus requires powders, lotions or ointments.

In general the amount of dressing applied in skin diseases should be the absolute minimum. Thick layers of lint and cotton wool are very bad because they cause so much heating of the part resulting in increased blood supply, exudation of serum and irritation. Ointments and pastes should be covered, if at all, with gauze or butter muslin and the minimum of bandage. If the patient is up and about pieces of old cotton

or linen may be pinned or tacked inside the underclothes over the affected part, or old, light-coloured or white, nylon, silk or rayon stockings with the feet cut off may be used to secure dressings on the legs and arms.

### Antibiotics

Penicillin is useful in certain infections of the skin due to staphylococci, streptococci and pneumococci, though resistant strains of staphylococci are becoming very common. Less susceptible are the gas-gangrene group *C. diphtheriae*, *B. anthracis*, *Corynebacterium rubeopallidum*, streptococcus *viridans* and actinomyces. Individual strains of the two latter may be resistant. Highly susceptible are the gram-negative cocci, gonococci and meningococci. *Spirochaeta pallida* is also sensitive. The following are resistant—the typhoid-dysentery-coli group streptococcus *faecalis*, *B. proteus* and *Pa. pyocyanea*, the tubercle bacillus, most of the filterable viruses, the ringworm fungi and the moniliae.

Penicillin is usually given by intramuscular injection into the upper buttock or outer thigh as follows—300 000 units of crystalline procaine-benzyl penicillin in 2 c.c. of sterile distilled water once daily or 500 000 units of crystalline benzyl penicillin in 2 c.c. of distilled water twice daily. The procaine-penicillin causes a slower rise and a slower fall in the penicillin in the blood stream than does the plain salt. The injections should be continued for two or three days after the infection appears to have been cured. Penicillin may be given equally effectively by mouth in the form of phenoxymethyl penicillin (penicillin V). Each capsule contains 125 mg. equivalent to 100 000 units of penicillin G. One capsule is given four hourly the last two doses being given together at night. It is best given after meals. Half the adult dose is sufficient for children and a quarter for infants.

Penicillin should not be used topically.

REACTIONS TO PENICILLIN—see page 128.

Tetracyclines (Achromycin) chlortetracycline (Aureomycin) and oxytetracycline ("Terramycin") are primarily

bacteriostatic drugs, as opposed to penicillin, which is bacteriocidal. They are broad-spectrum antibiotics, being effective against Gram positive and Gram negative bacteria as well as some of the rickettsiae and viruses. They should be reserved for infections which are resistant to penicillin. Tetracycline is the least toxic and is the member of the group usually employed. They are all given in capsules containing 250 mg. or as elixir for children, 1 gm. daily for 5 days being an average course for an adult.

Locally they are the antibiotics of choice for haemolytic streptococcal and for long standing chronic staphylococcal infections. For most purposes the  $\frac{1}{2}$  per cent ointments are as effective as the 3 per cent ones. Sensitivity reactions are uncommon.

Erythromycin has a similar antibacterial range to penicillin, but there is no relationship between the two or between it and the tetracyclines. It can thus be used when these other antibiotics are contraindicated either because of sensitivity in the patient (especially to penicillin) or resistance in the organism (particularly in staphylococci). Unfortunately staphylococci quickly become resistant to erythromycin. It is given orally in divided doses up to 2 grammes daily in adults. A 1% ointment is available.

Streptomycin is especially useful in infections by the tubercle bacillus, e.g. resistant cases of lupus vulgaris. The usual dose is 0.5 gm. intramuscularly twice daily for periods up to three months. Doses totalling more than 1 gm. a day are likely to be followed by sensitization phenomena and toxic effects on the eighth cranial nerve leading to tinnitus and vertigo.

**PRECAUTIONS** Rubber gloves should be worn when handling streptomycin as it readily causes sensitization of the skin.

Neomycin half per cent in ointment or lotion is the local antibiotic of choice in acute superficial infections due to staphylococci and gram negative organisms. It is not used systematically so that emergence of resistant organisms is a less serious occurrence. Further it very rarely causes a sensitivity reaction.

Polymyxin is also used topically. It is active against almost

all Gram-negative bacteria, but is employed especially for its effect on *Pseudomonas pyocyanea*. It is available combined with bacitracin in the form of "Polyfax" ointment.

Mycostatin (Nystatin) isolated from a streptomyces, is the only antibiotic to have any action against yeasts or fungi. It is used topically as a pessary suspension or ointment (100 000 units per gram or m.l.) to treat moniliasis of the mucous membranes and skin. Tablets containing 500 000 units are available for treating systemic moniliasis.

### Side Effects of antibiotics

All antibiotics if taken by the mouth and if given for more than a few days, are liable to cause stomatitis, black tongue and pruritus of anus and vulva. These have been ascribed to destruction of the normal bowel flora and consequent loss of the B group vitamins produced by them, and also to multiplication of other organisms such as *B. coli*, proteus, *Pseudomonas* and *Candida albicans*, due to removal of competing bacteria. Cases of fatal gastro-enterocolitis have occurred due to coagulase-positive staphylococci.

It is wise, therefore, not to use antibiotics unless driven to do so not only on account of their expense but also because of their side-effects, which can be very inconvenient and even dangerous to the patient.

Large doses of the B group vitamins should be given with the broad-spectrum antibiotics, and three or four cupsful of yogurt daily have been recommended.

### Cortisone and ACTH

Cortisone acetate supplements the natural supply of adrenal cortical hormone, while adrenocorticotrophin (ACTH) stimulates the adrenal cortex to turn out more of the hormone. How this hormone works no one knows at present, but it has the effect of suppressing the symptoms of many diseases, though it cures none. Because it does suppress the symptoms, it is essential that the patient's condition should be fully and correctly diagnosed before either of these substances is given,

or disaster may follow. Moreover if cortisone is supplied to the body the adrenal then makes less, and if the supply is suddenly stopped, the body is deprived of an adequate supply for some days. Conversely if ACTH is suddenly stopped the pituitary which has been having its work done for it, takes some days to make ACTH again and the adrenal, in consequence, fails to supply enough cortisone. It is therefore essential before it is decided to treat any patient with either of these preparations, to make sure that enough is available not only for the treatment but for the prolonged tailing-off that will be required otherwise the last state of the patient may be worse than the first.

Withdrawal symptoms are weakness, lassitude, anxiety and sweating. Occasionally the substitution of cortisone for ACTH or vice versa at the end of a course will reduce the symptoms of withdrawal.

Cortisone acetate is given by mouth as 25 mg tablets six hourly starting with a total daily dosage of 75-200 mg or less, though 400 mg or more may be required in pemphigus. The dose is reduced in stages by 12.5-25 mg every two or three days, but as rapidly as possible consistent with the control of symptoms, down to a maintenance dose of 25-50 mg daily.

Prednisone and Prednisolone derivatives of cortisone and hydrocortisone respectively are also active when given by mouth the equipotent doses being about a quarter to a fifth that of cortisone. They cause less water retention than does cortisone but in all other respects behave in the same way. They are the drugs of choice in dermatology and are available in one and 5 mg tablets.

Side-effects usually reversible, due to too high or (more commonly) too prolonged dosage, are deposits of fat ("moon-face" "buffalo-hump" supra-clavicular pads) oedema (often preventable by restricting common salt and giving potassium chloride, 1 ' gm daily), hirsutism, acne striae atrophicae (not reversible) skin pigmentation menstrual disturbances, osteoporosis (sometimes preventable by giving androgen as well as cortisone) psychic changes such as euphoria and elation and occasionally mental depression or mania in those predisposed to

psychoses. Complications of cortisone treatment are peptic ulcer and perforation, thrombo-embolic accidents, diabetes, miliary tuberculosis and aggravation of infections with staphylococci, streptococci, meningococci or pneumococci.

**Indications.** The only absolute indication for the use of these drugs is in pemphigus and pemphigoid, severe systemic lupus erythematosus, and exfoliative dermatitis and erythrodermia. These conditions may be fatal and do not respond to other therapy. Apart from these, however there are a number of other maladies, which can be suppressed wholly or partly by steroid hormones. They are not, however best so treated on every occasion and each case must be judged on its merits. Further other more routine methods of treatment must be continued even when these drugs are given. These conditions include sensitization dermatitis, drug eruptions, urticaria, most types of constitutional eczema, lichen planus, sarcoidosis, cutaneous lesions of the reticuloles, polyarteritis nodosa, dermatomyositis, generalised pruritus of unknown cause and thrombocytopenic purpura.

**Contra-indications** are cardiac failure, chronic renal failure, infection uncontrolled by antibiotics, peptic ulcer or past history of peptic ulcer haematemesis or melasma and past or present mental instability.

**ACTH** a purified protein fraction of the anterior lobe of the pituitary gland of cattle or pigs, is given as a solution in normal saline four times daily by intramuscular injection or once daily if incorporated in a gelatine base. The starting dose is 80-100 mg daily or in pemphigus 160-240 mg. daily. A more economical method is by intravenous drip in 500 c.c. 5% glucose saline running at 10 drops per minute, in which case 10-20 mg. is equivalent to 100 mg by intramuscular injection. 10 mg of heparin may be added to prevent venous thrombosis. The dose must be reduced to a maintenance dose as quickly as possible consistent with control of symptoms, just as in the case of cortisone.

**Side-effects, Indications and Contra-indications** of ACTH are similar to those of cortisone.



Hydrocortisone used as an ointment in a greasy or water soluble base or as a lotion, in  $\frac{1}{2}$  1 or  $2\frac{1}{2}$  per cent strengths, is an extremely useful local application. It is effective in many cases of eczema and dermatitis, seborrhoeic dermatitis, otitis externa and pruritus ani and vulvae. It must be stressed however that its action is only suppressive and that final cure must depend on the eradication of the primary cause. Recently it has become available combined with antibiotics, such as neomycin and the tetracyclines, and thus can be used in conditions such as infective seborrhoeic dermatitis, otitis externa, retro-auricular intertrigo and secondarily infected eczema and dermatitis.

Hydrocortisone can also be used as an injection, in the strength of 25 mg per m.l. and has been found useful in certain cases of verrucous lichen planus and keloids.

Physical methods of treatment have a wide field of application in dermatology. They may be classified as follows:

- 1 Cold CO<sub>2</sub> snow liquid oxygen or nitrogen.
- 2 Heat Galvano-cautery Paquelin's cautery and Diathermy
- 3 Electricity Galvanic baths, Electrolysis, Ionisation.
- 4 Radiation. Ultra violet light, Grenz rays, X rays, Radium Thorium X.
- 5 Skin Planing

Some of these methods, such as the use of CO<sub>2</sub> snow the cauteries thorium X and some of the uses of ultra violet light, come within the range of the general practitioner whereas others, such as Diathermy X rays and Radium and skin planing, require either more expensive apparatus or more skill and experience in their application than the general practitioner is likely to have time or inclination to acquire.

### Cold

CO<sub>2</sub> snow is made by allowing the liquid to escape from a cylinder when, owing to its expansion and consequent cooling, some of it is solidified into snow. This is caught in a cloth chamois leather or tube of blotting paper and is then compressed in a metal or vulcanite cylinder to form a solid stick. This stick is shaped by pressure between two metal

surfaces, e.g. a metal spatula and a hot water tap till it is of a size to fit the lesion to be treated.

Another method is to dissolve the loose snow in acetone until it forms a semi-solid slush which can be painted on the lesion. In my experience by far and away the best apparatus for the use of  $\text{CO}_2$  snow is the "Cryocautery" of Lortet-Jacob made by Drapier 41 Rue de Rivoli Paris. In this the  $\text{CO}_2$ -acetone slush is contained in shaped copper cones of various sizes which can be applied with any desired degree of pressure. Its advantages are an immense saving of time and of  $\text{CO}_2$ , and the provision of a point whose shape and size do not alter during the application.

The application of  $\text{CO}_2$  snow is only slightly painful and no sort of anaesthetic is required. Its action is to produce a local frost-bite which is succeeded by oedema, the formation of a blister thrombosis of vessels, with consequent necrosis and shedding of the frozen tissue, if the application has been sufficiently severe. After slight applications, e.g. in capillary naevi the thrombosis of dilated superficial vessels is the object aimed at and the necrosis is only minimal.

To freeze a common wart with  $\text{CO}_2$  snow a pencil of snow must be made and shaped so that it exactly fits the top of the wart. It must have approximately parallel sides so that the shape and size of the applied end will not alter appreciably as the snow evaporates where in contact with the wart. The snow must be compressed as hard as possible so that firm pressure may be applied to the pencil without causing it to alter its shape and size too quickly. A wart is much more easily frozen if it is first softened by allowing it to soak up drops of liq. potassium placed on it with a wooden applicator at intervals, for a quarter of an hour beforehand the softened keratin being rubbed away. Or immediately before applying the snow the wart may be moistened with acetone. The pencil of snow is applied to the top of the wart with firm pressure care being taken to keep it perpendicular to the skin surface and not to allow the sides of the pencil to make contact with the skin if the latter becomes depressed into a pocket by the pressure. The application should be continued until a rim

of frozen tissue a millimetre or so wide has appeared all round the base of the wart without this part having been in actual contact with the snow. When this appears it indicates that the freezing process has extended sufficiently deeply and the snow should be removed. The wart is now flat or depressed and yellow white in colour. In a few minutes it thaws and swells up to rather more than its previous size. In a few hours a blister may appear beneath it, lifting it up. Unless the blister is inconveniently large it is probably best not to open it. If large it may however be pricked aseptically and the fluid allowed to escape. In a few days it will dry up and the wart will eventually fall off in from ten days to three weeks. Often a wart will fall off after three weeks although no obvious blister ever formed. Very thick warts may require several freezings at intervals of two weeks or so before they are completely removed.

The time for which a pencil of  $\text{CO}_2$  must be applied varies enormously according to the character of the lesion under treatment. For lupus erythematosus, small superficial naevi and ordinary cavernous naevi in babies 10-30 seconds may be sufficient. For warts 2-4 minutes or even longer until the rim of frozen tissue described above appears all round the wart.

After freezing, the lesion should be left alone and kept dry being protected from friction if necessary until it has dried up into a scab.

The slush made by dissolving  $\text{CO}_2$  snow in acetone can be used in lupus erythematosus where a large area has to be superficially frozen. It can be painted on the lesions or spread on them like butter with a wooden spatula. It freezes the skin very quickly but superficially. Personally I am not fond of it as it is not well under control and pieces are apt to get into undesired places, such as down the patient's neck.

Liquid oxygen or nitrogen have a greater freezing effect than  $\text{CO}_2$  snow and only have to be applied for 20-30 seconds for the average wart. They are painted on with a pledget of cotton wool wrapped round an orange stick. They represent the best

method of cryotherapy in a hospital clinic where a large number of cases may have to be treated. Their drawback is that they have to be collected each time from the manufacturer since due to evaporation, a pint only lasts about 48 hours, even if kept in a thermos flask in a refrigerator

### Heat

The most convenient form of cautery for use in dermatology is the **galvano-cautery** the burner of which consists of a fine platinum wire point joining the ends of a pair of stout parallel copper wires. The point is heated by an electric current derived either from the mains (through a voltage-reducing device) or from an accumulator. The burners used in dermatology should be short, never more than  $1\frac{1}{2}$  inches long. The very long burners used in intranasal work, for which most cauteries appear to be designed, are inconvenient and difficult to control. The platinum point should be bent to form almost a right angle with the copper wires. A burner with a flattened leaf like point in line with the copper wires is useful as a knife for cutting off excrescences such as *condylomata acuminata*.

The best method of using the cautery is at a bright yellow heat for a fraction of a second at a time making repeated applications until the object is accomplished. Used in this way the destruction is quick and almost limited to the part in contact with the burner as the heat has not time to penetrate. The pain produced by this type of application also is less than that caused by applying a cooler burner for a longer time.

The uses of a galvano-cautery in dermatology are very numerous. For stellate and capillary naevi, telangiectases small warts or moles, and puncturing lupus nodules, it can be used without an anaesthetic. Under general or local anaesthesia it is invaluable for cutting off structures which bleed readily such as *granuloma pyogenicum* or *condylomata acuminata*. At a full red heat it is useful for sealing bleeding points in minor skin surgery.

*Pagelin's cautery* has the advantage of being self-contained and requiring no electricity. Its burner consists of a hollow

platinum point kept hot by blowing benzol vapour into it after a preliminary heating with a spirit lamp.

The various applications of high frequency oscillating electric currents for diathermy coagulation, and cutting while of some use in dermatology hardly come within the range of the general practitioner. With the exception of their use for the excision of malignant growths equally good results can be obtained by other and simpler means such as the galvano-cautery and electrolysis.

### Electricity

Galvanic hand or foot baths are very useful in the treatment of chilblains electrolysis for the removal of superfluous hairs, small moles and angiomas and ionisation for the removal of obstinate warts. For details a larger book on skin diseases or one on medical electricity should be consulted.

### Radiation

The uses of radiant energy in dermatology are very numerous and important. The forms used are ultra violet light, X rays, thorium  $\lambda$  and the beta and gamma rays of radium. Very soft X rays, the so-called "grenz rays," are also used by some dermatologists.

**Ultra violet light.** For details of the apparatus used to produce ultra violet light, and the technique of its application, reference should be made to works dealing specially with this subject. There are three principle sources, the carbon arc, fluorescent phosphor arc and the mercury vapour. Where general irradiation is needed the first two are of greater value but where greater skin stimulation is required locally i.e. in acne vulgaris and alopecia areata, the mercury vapour arc is better. For very localised treatments with contact the mercury vapour Kromayer lamp is used. This allows direct contact with the skin through a quartz window. Suitable quartz rods may be attached for treating sinuses or other small deep areas.

The effects of ultra violet light on the skin are to produce after a latent period of four to eight hours, an erythema which is an inflammatory reaction. This is followed if the erythema has been strong enough by visible peeling and later by pigmentation.

in the basal layer. The mercury vapour and carbon arc produce a greyish colouring, the fluorescent phosphor a more natural brownish. After a few days there is an increase in the thickness of the corneal layer of the epidermis and, to a lesser extent, also in the prickle-cell layer.

Definite degrees of erythema can be obtained and therefore ordered, as the inflammatory reaction can be accurately estimated.

*First Degree Erythema.* (E. 1) After about eight hours a gentle erythema is seen. This fades within twenty-four hours and is not followed by visible peeling.

*Second Degree* (E. 2) After about six hours, a marked redness with some irritation occurs. This will last from two to three days and is followed a day or two later by a fine powdery desquamation which continues over a further period of four to five days.

*Third Degree* (E. 3) This is a strong reaction which starts after a few hours and continues to develop for up to twenty-four hours. There is great redness, swelling and pain lasting for three to four days followed by a flaky desquamation the whole process usually being finished in another three to four days.

*Fourth Degree* (E. 4) This is a blister dose—the maximum erythema obtainable on skin.

The reaction of different individuals to the same lamp varies considerably and all treatment should be preceded by a skin test.

The thickening of the skin and the pigmentation appear to be protective in function. The important effects of ultra violet light are that they appear to stimulate growth and raise the resistance of the skin to infection, as well as causing formation of vitamin D from the sterol in the epidermis or sebum thus indirectly affecting the calcium and phosphorous content of the blood. A course of small doses to large areas of the body cause a feeling of well being, but this may be subjective. If given in strong doses ultra violet radiation can be powerfully bactericidal.

While ultra violet light is actually falling on the skin it causes fluorescence that is, it makes the skin give out visible light of a longer wave length than the invisible ultra violet light falling on it. Such fluorescence is of low intensity and can therefore

only be seen in a dark room. Steps must be taken to cut out of the beam from the lamp all the visible light and only allow the ultra violet to come through. This is achieved in practice by using an enclosed arc such as a mercury vapour lamp or tungsten arc and filtering the emergent beam through a plate of Wood's glass. This is a dark violet glass containing oxide of nickel, and transmits only the long ultra violet rays of wave length from about 3800-3300 Ångstrom units together with a very little red and visible violet. Normal hair and skin fluoresce only very slightly under this light, looking as though they had been dusted with flour the finger nails fluoresce rather more brightly but hairs infected with the common small-spored ringworm fluoresce brilliantly with a greenish light. This effect is used in diagnosis (see Roxburgh A. C. *Brit Jour Derm & Syph* 1927 39 361 and 358).

FOR TREATMENT ultra violet light is used in dermatology both locally in large doses to small areas, and generally in small doses to large areas of the body. It is also used with pressure for its caustic effects. The various effects of ultra violet light are used in dermatology as follows:

1 Suberythema or very mild E. 1 doses, given generally are of value in such conditions as seborrhoeic dermatitis or as a general tonic. In certain mild cases of eruptions caused by light, they may be used to desensitize.

2 General E. 1 are given for psoriasis, multiple boils and pityriasis rosea.

3 Local E. 1 for certain cases of pruritis.

4 The stronger irritant effect of an E. 2 is of value in acne vulgaris and alopecia areata.

5 An E. 3 may be used for smaller patches of alopecia areata, or as a counter irritant.

6 The bactericidal and stimulating effect of very strong doses make ultra violet radiation useful in the treatment of infected or indolent wounds and ulcers.

7 The destructive effect may also be used with pressure from a Kromayer lamp in lupus vulgaris.

Although ultra violet light is of great value in dermatology

particularly in the conditions mentioned above if the glowing accounts of cures by its means of an enormous variety of different skin diseases, such as are detailed in some works on ultra violet light therapy are taken at their face value disappointment is likely to result.

Skin diseases in which ultra violet radiation is contra indicated are acute dermatitis and eczema, acute psoriasis and lupus erythematosus. It is also unwise to give it in conjunction with injections of heavy metals such as gold, and in patients with pulmonary tuberculosis. It should also be borne in mind that patients with diabetes, or those receiving such drugs as the barbiturates, are sensitive to ultra violet light and their doses must be adjusted accordingly.

**X rays** Although  $\lambda$  ray treatment hardly comes into the sphere of the general practitioner yet it is of such immense value and of such wide application in dermatology that it is desirable that he should know in general the effects of  $\lambda$  rays on the skin and the diseases in which they are likely to be of use. Also their possible ill effects and contra indications.

In general, the effect of  $\lambda$  rays appears to be to reduce the activity of cells in all their various functions; to have in fact an "ageing" effect upon them. The reproductive function of the cells that is their power of division, is one of the first to be affected.

**MEASUREMENT OF  $\lambda$  RAYS** This depends on their ionising properties and is expressed in Roentgen or "r" units. By means of a small condenser known as an ionisation chamber apparatus can be calibrated directly in r units. The penetration and ultimate effect of  $\lambda$  rays also depends on their quality. Rays of long wavelength which are produced by a low kilovoltage are easily absorbed and thus are useful for superficial irradiation. Short rays, on the other hand produced by a high kilovoltage are penetrating and constitute deep  $\lambda$  ray therapy. Thus the types of X rays used in dermatology are

- 1 Grenz rays (7-10 kV) which exert their effects almost entirely on the epidermis and dermis.

2. Contact  $\lambda$  rays (30-60 kV) (Chaul tube therapy) in



which the beam traverses the lesion and which are used for superficial neoplasms.

3 Superficial X rays (10-140 kV) which are used for the bulk of dermatological conditions

The CLINICAL USES of X rays may be classified as depending upon the following effects which they have upon the tissues.

1 Cessation of cell division and death of cells. Doses of 400-1200r. Used in treatment of new growths and warts and to produce permanent epilation.

2 Reduction of rate or temporary cessation of cell division. Doses of 400r or smaller doses repeated. Used to produce temporary epilation reduction of sebaceous secretion in acne, reduction of scaling, hyperkeratoses and corns. Reduction of cellular infiltration in eczema lichen planus, lichenification, and chronic inflammations generally. Reduction of young angiomata and granulation tissue. Flattening of hypertrophic scars.

3 Reduction of secretion. Doses of 50r-300r repeated. Used in a ne localised hyperhidrosis and weeping eczema.

4 Reduction in sensitivity of nerve endings. Doses of 50r-200r. Used in pruritus eczema, lichen planus, lichenification.

5 Modification of tissues rendering them less suitable for bacterial growth. Doses of 23r 100r. Used in furunculosis, styes, streptococcal fissures, and chronic paronychia.

6 Desensitisation of the epidermic cells. Doses of 50r 100r. Used in eczema and dermatitis.

It comes to this, that owing to the above effects singly or more usually perhaps in combination there are but few chronic diseases of the skin which cannot be improved in greater or less degree by X ray treatment. Even in some relatively acute conditions such as weeping eczema small doses are beneficial.

For non malignant conditions doses of 50r may be repeated if necessary in four days 100r in seven days 200r in two weeks, 300r in three weeks and 400r in one month, but it is often wiser to lengthen or even to double these intervals. The total dose permissible to any one area of skin depends on the rate at which it is given, but I always regard 800r in three or four months as a maximum which should not be exceeded if this can be avoided.

After a year or two another 400r may be given in divided doses if necessary but I try to make a total of 1200r an absolute maximum for any period of time. I have however seen cases where much more has been given with perfect safety over a period of many years but there the doses never exceeded 400r and the intervals were measured, usually in months.

**DISADVANTAGES OF X RAY TREATMENT** X rays cause some degree of pigmentation of the exposed skin which varies in different patients and is quite pronounced in some brunettes. This is sometimes a drawback from a cosmetic point of view but with a little skill in application to avoid sharp edges between the exposed and unexposed portions the pigmentation need not be very noticeable. The only important drawback to X ray treatment lies in the possibility of overdosage either by a single dose or by the summation of small doses. Therefore X ray treatment should only be carried out by careful and experienced workers. There is no room for carelessness or for slap-dash methods in the administration of X rays for treatment. The same applies to their use for diagnosis, for the two worst X ray burns I have ever seen were both the result of prolonged screening for observation of barium meals.

**CONTRA INDICATIONS FOR X RAY TREATMENT** The application of X rays, at least in doses approximating to 100r to areas of acute impetigo leads to a great extension of the eruption over the X rayed area. Therefore cases of ringworm of the scalp should not be X rayed unless or until the head is free from impetigo. Areas of lupus erythematosus should not be treated with X rays at all.

**Radium Radon and Radio-active Isotopes** The uses of these sources of radiation in dermatology are mainly in the treatment of rodent ulcers, squamous epitheliomata, cheloids, subungual warts and cavernous angiomata. As they should only be handled by those skilled in their use no description of methods or dosage will be given here.

**Thorium X** A form of radiation therapy easily applied by general practitioners is that by Thorium X. This is a degradation product of mesothorium which gives off 92% alpha 4%.

bets and 4% gamma radiation. Alpha rays are extremely active in producing ionisation in molecules through which they pass, with resulting changes in the tissue but as they hardly penetrate 0.3 mm (Lomholt) which is little deeper than the epidermis, they can be applied many times to the same area of skin without risk of any permanent damage thus differing from X rays. Correspondingly however they are much less effective than  $\lambda$  rays in the treatment of the majority of skin diseases. For a study of the penetration of the epidermis by Thorium X see V. H. Witten *et al. Jour. Invest. Derm.* 1951 17 311. Thorium X has been prepared for many years by the firm of Degos of Berlin. About 1939 I persuaded Messrs. Derby & Co. Ltd 11/12 St Swithun's Lane London, E.C. 4 to undertake its manufacture in this country and it is now obtainable by post by writing to them. It can be had either suspended in a pyroxylin varnish, in solution in propyl alcohol or in a eucerin ointment. It is usually used in a strength of 1000 to 2000 electrostatic units (e.s.u.) in 1 c.c. of the vehicle but can be had either stronger or weaker. The date and time at which it is to be used must be specified on the order because it decays at a rate reducing its output of alpha rays to half value in about 3.5 days, and to practically nothing in 10 days. If Thorium X is painted on the skin say in a strength of 1500 electrostatic units in 1 c.c. varnish and allowed to dry no immediate effect will be seen. After a day or two an erythema will appear under the varnish which goes on increasing in intensity for several days and is accompanied by slight oedema. The latter soon subsides, but the redness persists for two to three weeks. Then it gradually fades with some slight desquamation and is replaced by pigmentation. This remains for many weeks but also eventually disappears. Histologically according to Lomholt, the epidermis undergoes degeneration which reaches its maximum in two weeks, after which there is less change but the type of cell appears inflammatory in character.

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A wooden applicator is the best thing with which to apply the varnish. With a wisp of cotton-wool wound round it this does well also for the alcoholic solution.

If using an alcoholic solution 1000 e.s.u. per a.c. if using varnish or ointment 1500 e.s.u. per a.c., are usually sufficient for most purposes. The effect of the alcoholic solution can be increased by varnishing the painted area with collodion as soon as the alcohol has dried. It is important that all scales, ointment etc. should be removed from the skin to be painted, and that the painting should be limited to the area required, because of the succeeding erythema and pigmentation. The painting is repeated if necessary when the redness has quite subsided, the interval depending on the type of lesion being treated and on the strength of Thorium X applied.

I have found Thorium X very useful in some cases of patchy eczema pruritus ani andichenification but especially in two conditions which are otherwise most difficult to treat, viz. verrucoe lichen planus and capillary naevi in babies. For the majority of skin conditions for which one requires radiation such as eczema, acne, ichenification, cheloids, pruritus ani and vulvae and chronic patches of psoriasis, I find X rays much more effective.

### SKIN PLANING

Skin planing, or dermabrasion, is used mostly for the removal of acne scars, but it has proved beneficial in certain carefully selected cases of smallpox and chicken pox scars, accidental and intentional tattooing, keloids and hypertrophic traumatic scars, and vascular naevi. Freon spray (chemically related to ethyl chloride) is the anaesthetic used, since it produces rigidity of the skin, together with haemostasis abrasion being produced by a power-driven steel wire brush. Care is taken not to descend to the subcutis, epithelialization taking place from the remnants of the sweat glands and pilo-sebaceous follicles.

Skin planing is a delicate and intricate operative technique and should only be undertaken by experts, in suitable surroundings and with adequate equipment.

## CHAPTER IV

### CONGENITAL AFFECTIONS OF THE SKIN

#### ICHTHYOSIS

**VARIETIES.** *Xeroderma* (mild type) *Ichthyosis simplex* (ordinary type) *Ichthyosis foetalis mitior* *Ichthyosis foetalis gravior* (harlequin foetus)

**DEFINITION** An abnormality of congenital origin appearing at birth or in the first year and characterised by dryness, roughness and scaling of the skin.

**ETIOLOGY AND PATHOGENESIS.** The cause is unknown but has been ascribed to some defect in the thyroid secretion. The disease is often hereditary.

**PATHOLOGY.** Hyperkeratosis, decrease of the prickle-cell and granular layers and flattening or sometimes hypertrophy of the papillae. Sebaceous glands small and few. Sweat glands usually normal.

**CLASSIFICATION** All degrees of the disease occur from a slight dryness of the skin with branny scaling (*Xeroderma*) up to the condition known as "Harlequin Foetus" (*Ichthyosis foetalis gravior*) in which the child is born prematurely and appears to be encased in an armour of horny plates two or three millimetres in thickness. Such infants are either still born or only survive a few days.

**SIGNS AND SYMPTOMS** In the slight cases (*Xeroderma*) there is dryness and slight scaling of the skin. The minute furrows of the skin surface are exaggerated and the horny layer is apt to crack along these lines. On the buttocks and the extensor surfaces of the limbs there may be hyperkeratosis at the mouths of the hair follicles (*Ichthyosis follicularis*) (p 53) causing a sort of permanent "goose skin" appearance.

In rather more severe cases (*Ichthyosis simplex*) (Fig 1) there is



the desquamation following scarlet fever the skin may appear normal for a time but the scaling soon returns.

**DIAGNOSIS** This is usually easy. The history dating from early life the family history the general dryness and scaling of the skin worst on the extensor surfaces, the fact that the flexures are normal and that the patient is better in hot weather are the points to which attention should be directed.

**PROGNOSIS** The disease will probably increase up to puberty

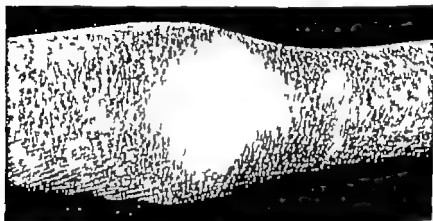


FIG. 1.

Ichthyosis Simplex. Note normal skin in antecubital fossa.

after which it will remain stationary. It is not curable but treatment can ameliorate it somewhat.

**TREATMENT INTERNAL.** Vitamin A, given in doses up to 400 000 units daily has a beneficial effect but only causes improvement while it is being given.

**EXTERNAL.** This is the most important. The scales should be removed daily by warm baths with bran or starch (p. 26) using a super-fatted soap. After drying the skin should be lubricated with 20% to 50% glycerine in water or with olive oil almond oil or emulsions of these with equal parts of lime water or else with equal parts of Ung. ac. salicyl. (2%) and Glycerin amyli. I have found ordinary petroleum jelly as good as anything in most cases. Ichthyotic patients should wear warm clothing and avoid cold winds.

Twice weekly generalized ultra violet light for 2 or 3 months is also frequently helpful.

# Ichthyosis Follicularis

**SYNONYMS.** Keratosis supra follicularis, Keratosis pilaris.

**DEFINITION** A condition of the skin, congenital and familial, characterized by the presence of horny plugs filling the openings of the hair follicles and projecting above the surface as acuminate papules.

**ETIOLOGY AND PATHOGENESIS** It appears to be a variety of ichthyosis in which the follicles are more affected than the intervening skin (MacLeod)

**PATHOLOGY**(MacLeod) Dilatation of the mouths of the hair follicles, which are filled with horny plugs extending above the level of the skin. The lower two-thirds of the follicle are atrophied and contain atrophied hairs. Sebaceous glands atrophic or absent. These atrophies are apparently due to the pressure of the horny plug. There is slight dilatation of vessels and infiltration of small round cells in the corium, also probably secondary to the pressure of the plug.

**SIGNS AND SYMPTOMS** The condition is usually first noticed at two or three years of age. In the mildest degrees, which are much the most common, it amounts to little more than a permanent prominence of the follicles on the extensor aspects of the limbs. In the more severe cases the condition is widespread including the face, but the extensor surfaces of the limbs are the most affected. The skin is dry, scaly and harsh, and feels like a nutmeg grater. The horny plugs can be picked out and leave conical depressions. Eventually the plugs fall out of themselves and leave small depressed scars.

In the most severe cases the whole body is affected, except the palms and soles and the hairs of the scalp, eyebrows, and lashes are absent.

**DIAGNOSIS.** From *goose-skin*. The latter is transient and due to cold or fright. The hairs are normal, and the papules are only the mouths of the follicles made prominent by erection of the hairs.

From *Lichen spinulosus*. The latter consists of circumscribed patches a few inches in diameter covered with prominent follicles each surmounted by a filiform spine. The hairs are not atrophic



as in *Ichthyosis follicularis* and the spine consists of concentric horny lamellæ surrounding the hair (Adamson). *Lichen spinulosus* is most common on the neck, buttocks and extensor surfaces of the limbs in children, and is easily cured by soap and salicylic acid. The rest of the skin is normal and the condition is not familial or congenital.

From *Lichen scrofulosorum* (p. 316). The latter consists of circumscribed groups of prominent follicular papules, usually on the trunk in children who have tuberculosis.

From *Lichenoid trichophytiæ* (p. 179). The latter is a widespread eruption of follicular papules occurring in patients with deep-seated ringworm.

From *Lichen plano-pilaris* (p. 366). This is a variety of *Lichen planus*, in which some of the papules are acuminate and follicular instead of being flat. It usually occurs in adult women.

From *Phryoderma* (Vitamin A. fatty acid deficiency) (p. 461). This comes on suddenly is accompanied by other signs and is rapidly cured by giving Vitamin A. and essential fatty acids.

From *Scurvy* (p. 463).

From *Pityriasis rubra pilaris* (p. 469). In this disease the papules tend to be reddish brown, they are nearly always present on the backs of the proximal phalanges of the fingers, and are usually associated with large areas of red scaly skin on parts of the body and limbs, with thickening of the nails and of the palms and soles.

From *Darier's disease*. The latter is very rare and is characterised by the presence of symmetrical areas covered with dark brown horny papules tending to coalesce, and in the later stages by vegetations in the flexures.

PROGNOSIS. *Ichthyosis follicularis* tends to persist indefinitely.

TREATMENT. Removal of horny plugs with hot water and soap, and applications of salicylic acid ointment 2 to 4%.

## TYLOSIS

SYNONYMS. *Keratoma palmare et plantare hereditarium*. *Hyperkeratosis congenitalis palmaris et plantaris*.

DEFINITION. A hereditary and familial hyperkeratosis of the palms and soles.

**ETIOLOGY AND PATHOGENESIS.** Unknown.

**PATHOLOGY** An enormous hyperkeratosis of the palms and soles usually without other changes

**SIGNS AND SYMPTOMS** The skin of the palms and soles is very thick and usually yellow in colour the thick yellow area coming to an abrupt end on the side of the hand or foot. Immediately beyond the edge of the yellow area there is sometimes a narrow band of erythema. The thickened horny layer sometimes cracks along the lines of the principal furrows, and such cracks may become infected. In some cases there is an associated hyperhidrosis which keeps the thick horny layer moist and sodden. The disease is usually first noticed at about four years of age and persists throughout life

**DIAGNOSIS** The symmetrical affection of both hands and feet the history and the absence of eruptions elsewhere on the skin should make it easy to distinguish this disease from hyperkeratosis due to hard work from arsenical hyperkeratosis (p 119) and from hyperkeratosis due to eczema (p. 332) psoriasis (p 345) or pityriasis rubra pilaris (p 54).

**PROGNOSIS.** The condition is incurable

**TREATMENT** Temporary improvement can be obtained by the application of strong salicylic acid ointments, 10 to 20%, and the subsequent use of soft soap and hot water to remove the softened horny layer. Small repeated doses of X-rays are of use also especially in securing the healing of the cracks.

## ALBINISM

**DEFINITION** A congenital absence of pigment from the skin eyes, and hair

**ETIOLOGY** Its cause is unknown. It is occasionally hereditary and familial.

**CLASSIFICATION** Albinism is usually complete partial cases being rare. When they occur the pigment free areas are arranged in a segmental or zoniform manner (Naevus achromicus)

**SIGNS AND SYMPTOMS.** Albinoes have a white skin, pink irises, and white or very pale yellow hair. Owing to the absence of protective pigment from the choroid they suffer from photophobia

and *hypstagnus* and owing to its absence from the skin they are readily "burned" by the sun.

**PROGNOSIS** The condition is permanent.

**TREATMENT** None is effective. Protection of the eyes by tinted glasses and protection of the skin from the sun (p. 79) will probably be required.

## NÆVI

**SYNONYMS.** Birth marks.

**DEFINITION** *Nævi* are localised abnormalities usually hyperplasias, of some elements of the skin or subcutaneous tissue of congenital origin. Exceptionally a *nævus* is due to a hypoplasia, e.g. *Nævus anemicus* *Nævus achromicus*.

**ETIOLOGY** Unknown. They are often hereditary. They frequently occur on sites corresponding to embryonic clefts and also at points where cutaneous nerves emerge from the deep fascia. They are sometimes associated with other developmental anomalies, such as hare-lip, cleft palate or webbed fingers.

**HISTOPATHOLOGY** This will be dealt with as the different types are considered.

**Classification of the commoner forms of Nævi**

Vascular nævi consisting of blood vessels.	Capillary hæmangioma (port wine stains)
	Cavernous hæmangioma (strawberry marks)
	Stellate hæmangioma ("spider nævus")
	Senile hæmangioma (Campbell-de Morgan Spot)
Vascular nævi consisting of lymphatic vessels.	Lymphangioma circumscriptum.
Non vascular nævi.	(1) Plane
	(2) Raised
	Pigmented maculae.
	(a) Cellular nævi, "Moles"
	Non-pigmented.
	Pigmented
	Hairy
	Giant.
	(b) Epidermal nævi.
	Verrucose
	Linear segmental.
	Ichthyosis hystrix.
	(c) Fibromatous.
	Neurofibromata
	von Recklinghausen's disease

**Capillary haemangioma.** **SYNONYMS.** Port-wine stain, Naevus flammeus.

**DESCRIPTION.** A localised area of the skin in which the superficial capillaries are dilated and more numerous than usual, giving



FIG. 2.

Capillary Naevus (port-wine stain). The black lesions are raised, purple cavernous angiomas of recent development.

it a pink, red or purple colour. The deeper blood vessels may also be involved. Typically the lesion is not raised.

**CLINICAL FEATURES.** Usually situated on the face and very common on the occipital region, but may be anywhere. Size from  $\frac{1}{2}$  to many inches across (Fig. 3). Usually noticed at birth or a day or two later.

**PROGNOSIS** Increases usually only with and in proportion to the growth of the child. If present at birth the naevus may disappear in a few months especially if situated at the root of the nose but if it persists after the first few months of life it will probably be permanent.

**TREATMENT** Probably the best treatment is to paint the naevus with Thorium  $\Lambda$  in varnish of a strength between 500 and 1500 a.s.u. per c.c. (p. 47). Five hundred might be used for the first treatment in an infant and 1500 in an adult. It is necessary to produce a definite inflammatory reaction which makes the naevus much more red than before. When this has completely subsided say in about a month probably the whole area will be somewhat less red than before treatment, though possibly somewhat pigmented and there may be islands almost free from redness. The remaining red areas may then be painted again, adjusting the strength of the varnish by the result produced by the previous painting. The process may be continued until a satisfactory degree of blanching has been achieved, or no further improvement takes place. Grenz rays (p. 45) and the local application of radio-active isotopes may also produce some fading. Freezing with  $\text{CO}_2$  slush or liquid oxygen or nitrogen for 10-30 seconds at monthly intervals has the same effect. No treatment however causes complete removal of the blemish and sooner or later a cover cream has to be employed. These are nowadays extremely effective and are capable of disguising the majority of lesions.

**Cavernous haemangioma.** **SYNONYM.** Strawberry mark.

**DEFINITION** A localised area of skin in which the capillaries are dilated tortuous and widened-out in places to form cavernous spaces like those in erectile tissue. The vessel walls are thickened.

**CLINICAL FEATURES** The lesions are usually raised and may be partly cutaneous and partly subcutaneous. When of this type there is a red patch of dilated vessels on the surface with around it a raised bluish area which represents the subcutaneous part of the naevus. Cavernous naevi are red or purple in colour and in size vary from a quarter to one or two inches in diameter. They are usually soft and compressible but this varies with the

amount of fat and fibrous tissue present. They vary in prominence with the venous pressure, being larger when this is raised, *e.g.* when the infant cries. Cavernous naevi are most common about the face and scalp but may occur anywhere. They may



FIG. 4.

Cavernous Naevus on forehead of 10 months old baby



FIG. 4a

Same Naevus four years later without any treatment.

be present at birth or may not be noticed till some weeks later

Occasionally they are injured and bleed freely but the bleeding is easily stopped by pressure. When situated about the vulva they often get excoriated and septic. The ulceration so produced frequently initiates a process of scarring which leads to the eventual obliteration of the naevus

**PROGNOSIS.** They usually increase in size for some six months then become stationary and the great majority disappear spontaneously by the end of the fifth year leaving either no mark at all or a trace only of atrophy or pigmentation. (Figs. 4 & 4A)

**TREATMENT** Active interference is rarely necessary. Sometimes, however if the lesion is particularly disfiguring or when painful ulceration occurs, as in vulval or perianal lesions, it is warranted. Radiotherapy either with X rays or radium, is the best method although the full effects are seldom apparent in under a year. Excision should be reserved for small lesions which will not need replacement grafts. Injections of sclerosing fluids, such as quinine urethane or boiling water can produce excellent results in expert hands. Freezing will only affect the most superficial lesions without causing scarring and is, therefore, seldom helpful.

**Stellate haemangioma** **SYNOXYMA** Spider naevus.

**DEFINITION** A small type of naevus consisting of a dilated central vessel bright red in colour and about one millimetre in diameter with smaller dilated vessels radiating from it for a few millimetres, and so suggesting the body and legs of a spider respectively.

**CLINICAL FEATURES** They commonly appear in childhood or in adult life, and it is probable that many of them are not born at all, i.e. they are not congenital defects, but should be regarded as acquired dilatations of vessels. They are commonest about the nose, cheeks and forehead and are only slightly disfiguring, but bleed freely if injured.

**PROGNOSIS.** They have no tendency to spontaneous disappearance but are easily removed by treatment.

**TREATMENT** The central vessel can be obliterated by a momentary stab with a fine pointed *galvano-cantery* at a bright yellow heat. Pressure should be applied for a few minutes thereafter followed by a drop of contractile collodion. In nervous children the cantery point may be applied cold the current having been adjusted to heat the point quickly. The switch in the handle is

then surreptitiously closed, the point heats up and the job is done before the child has jerked its head away.

In adults electrolysis is preferable for single lesions, being slower and more under control.

A fine point of  $\text{CO}_2$  snow can be used also for 30 seconds, but it is difficult to keep the point fine enough under the pressure of application and the Lortet-Jacob Cryocautery (p. 38) is preferable.  $\text{CO}_2$  snow is usually the best treatment in children.

**SEXILE HAEMANGIOMA**, Synonym. Campbell-de Morgan spot.

**DEFINITION** A small papular naevus, very common in persons past middle life consisting of a group of dilated capillaries.

**CLINICAL FEATURES** Small, raised, soft, dark red papules 1 to 5 mm. in diameter present frequently in large numbers, on the upper trunk of middle-aged and elderly individuals. They do not disappear spontaneously.

**TREATMENT** As for stellate haemangiomas.

## LYMPH VASCULAR NAEVI

**SYNONYM** Lymphangoma circumscriptum.

**DEFINITION** A comparatively rare form of naevus consisting of a group of vesicles in the corium formed by the dilatation of lymphatic vessels.

**CLINICAL FEATURES** Usually noticed at birth or in the first few months of life but may not appear until puberty or even later. The appearance is that of a patch of translucent white grey yellow or pinkish vesicles of firm consistence. There may be dilated blood vessels among them or some hyperkeratosis over them. They are most common on the neck, sides of the trunk, and on the upper limbs but occasionally occur on the tongue. Usually they cause no symptoms, but they have no tendency to spontaneous cure.

**DIAGNOSIS** The history and the absence of pain, inflammation or adenitis will distinguish a lymphangoma from a patch of *herpes zoster*.

**TREATMENT** Wide and deep excision is usually necessary to avoid recurrence. In small lesions  $\text{CO}_2$  snow (p. 39) electrolysis or cautery (p. 41) may be tried.



**PROGNOSIS** They usually increase in size for some six months, then become stationary and the great majority disappear spontaneously by the end of the fifth year leaving either no mark at all or a trace only of atrophy or pigmentation. (Figs. 4 & 4A).

**TREATMENT** Active interference is rarely necessary. Sometimes, however if the lesion is particularly disfiguring or when painful ulceration occurs as in vulval or perianal lesions, it is warranted. Radiotherapy either with X rays or radium, is the best method although the full effects are seldom apparent in under a year. Excision should be reserved for small lesions which will not need replacement grafts. Injections of sclerosing fluids such as quinine urethane or boiling water can produce excellent results in expert hands. Freezing will only affect the most superficial lesions without causing scarring and is therefore seldom helpful.

**Stellate haemangioma. Spitzoma. Spider naevus.**

**DEFINITION** A small type of naevus consisting of a dilated central vessel bright red in colour and about one millimetre in diameter with smaller dilated vessels radiating from it for a few millimetres, and so suggesting the body and legs of a spider respectively.

**CLINICAL FEATURES** They commonly appear in childhood or in adult life and it is probable that many of them are not seen at all, i.e. they are not congenital defects, but should be regarded as acquired dilatations of vessels. They are commonest about the nose, cheeks and forehead, and are only slightly disfiguring, but bleed freely if injured.

**PROGNOSIS** They have no tendency to spontaneous disappearance, but are easily removed by treatment.

**TREATMENT** The central vessel can be obliterated by a momentary stab with a fine pointed *galvano-cautery* at a bright yellow heat. Pressure should be applied for a few minutes thereafter followed by a drop of contractile collodion. In nervous children the cautery point may be applied cold the current having been adjusted to heat the point quickly. The switch in the handle is

## NON VASCULAR NAEVI



FIG. 5

Lymphangioma, Heerbrant. Note scars of previous attempts at removal.

Hairy moles are of either of the above types, i.e. pigmented or non pigmented. The hairs are usually strong (Fig. 6). Giant naevi are comparatively rare and consist of enormous

pigmented hairy moles involving large parts of the trunk. They are usually situated on the upper part of the trunk, the "fur cape" type, or about the buttocks, the "bathing-drawer" type. The affected skin is usually thickened darkly pigmented and thickly covered with strong hair which may be so profuse as to suggest an animal's coat. Such patients usually have very numerous small pigmented moles also.

**HISTOPATHOLOGY.** Moles consist of an aggregation of naevus cells, which are oval or cuboidal in shape with large vesicular nuclei. They originate from the dendritic cells (melanoblasts) of



FIG. 6.

Pigmented Hairy Mole. 8. Recently removed by plastic operation.

the basal layer of the epidermis. In dermal or resting moles, the cells are all in the dermis and are no longer capable of multiplying. Clinically they are raised, not markedly pigmented and frequently hairy. Junction naevi or moles (so called because the cells remain at the epidermo-dermal junction) are usually flat, black or dark brown, and hairless. In them the naevus cells are still



FIG

Segmental Naevus. Naevus unius lateris.

in the basal layer or just underneath it and are being actively formed. Mixed types occur but it is only moles with some junctional activity which are capable of becoming malignant.

The pigment in naevi is melanin, and is contained in the basal and prickle-cell layers of the epidermis, in chromatophores lying in the corium and in the naevus cells themselves.

**PROGNOSIS** Moles have no tendency to spontaneous disappearance. They usually grow slowly in size after their first appearance and then become stationary. Very rarely pigmented moles after middle life undergo a malignant change and begin to get darker in colour and to increase rapidly in size. The melanotic tumours so formed are exceedingly malignant.

**TREATMENT** Small moles may be removed by *electrolysis* or *excision*. The raised non pigmented moles which are often seen on the face can be satisfactorily removed by slicing them off level with the skin with a razor under a local anaesthetic, the bleeding being stopped by pressure with or without the help of adrenalin.

Large hairy moles on the face can sometimes be excised and the defect repaired successfully by a plastic operation. Moles which do not show and are not subject to friction or pressure are best left alone. Intensely bluish-black moles should either be left severely alone or else widely excised for it is this type which is most likely to become malignant. In the rare cases where a pigmented mole begins to grow rapidly or to become irritable it should be widely excised at the earliest possible moment either by scalpel or by diathermy the regional lymph nodes being removed at the same time.

(b) **Epidermal naevi.** These naevi are characterised by an overgrowth of the epidermis, especially of the horny layer and differ from the soft naevi in that there are no naevus cells or any characteristic changes in the corium. They may be of all sizes and degrees from groups of small warty lesions a quarter of an inch or so in diameter to black horny outgrowths involving a large part of a limb. They are usually noticed during the first year of life and increase with the growth of the child. They may be pigmented or non pigmented and are usually hairless.

**Verrucose naevus.** A patch of rough warty skin on the trunk or limbs.

**Linear naevus** (Segmental naevus, *Naevus unius lateris*) These consist of streaks or bands of irregular width occurring usually on one side of the body only although they may be bilateral. They often extend the whole length of the upper or lower or both limbs of one side, and are often found on the side of the trunk also. On the trunk they usually stop abruptly at the middle line. They may be composed of slightly raised horny papules, the colour of the normal skin, resembling plane warts or the lesions of lichen planus, or the lesions may be thicker and darker than this (Fig 7).

**Ichthyosis hystrix.** This is an extreme form of linear warty naevus in which the lesions are horny and dark brown or black in colour projecting as much as half an inch or more from the surface (Fig 8)

**DIAGNOSIS OF EPIDERMAL NAEVI.** This is not usually difficult if the history is kept in mind. *Plane warts* (p 235) *linear lichen planus* (p 365) *lichenification* (p 287) *warty lupus vulgaris* (p 207) are the conditions most likely to cause confusion.

**TREATMENT** In slight cases the lesions may be kept flatter and smoother by the use of salicylic acid ointment 2-10% and alkaline soaps. Small lesions may be removed by excision or destroyed by electrolysis, cautery (p 41) or diathermy. For the larger thicker lesions no treatment except excision, followed if necessary by skin grafting is likely to be of much use. Skin planing is ineffective, since it is always followed by regrowth of the lesion.

(c) **Fibromatous naevi.** *Neurofibromata* *Molluscum fibrosum* von Recklinghausen's disease.

*Neurofibromata* are soft tumours which may be of three kinds (a) occurring in the course of peripheral nerves, (b) forming projections on the skin surface (c) forming large pendulous masses. They appear to be developed either from the connective tissue elements in nerves, in which case they are of mesoblastic origin, or else (Verocay) from imperfectly differentiated embryonic nerve cells, in which case they are epiblastic.

**HISTOPATHOLOGY** The tumours are made up of what appears to be exceedingly cellular soft connective tissue. This, however, stains differently from normal connective tissue. Degenerated nerve fibrils are found in the tumours and elastic tissue is absent.

**CLINICAL FEATURES.** Neurofibromata occurring in the course of peripheral nerves are felt as small nodules under the skin. They are sometimes tender on pressure. Those which appear on the skin surface are soft, sessile or pedunculated tumours. On palpation they feel as though there were a hole in the skin under



FIG. 8.  
Ichthyosis Hystrix.

neath them through which they can be pushed. They vary in size from  $\frac{1}{4}$  to 1 inch or so in diameter and may be very numerous. The larger ones tend to be pedunculated. The large pendulous masses, when they occur hang in folds from some part of the body and may weigh many pounds.

**von Recklinghausen's disease** is a condition often hereditary and familial of which the three characteristic features are (1) pigmentation of the skin in the form of pigmented macules or patches, (2) neurofibromata on the peripheral nerves, (3) superficial neurofibromata sometimes forming large pendulous masses. The pigmented macules usually appear in childhood or early adult life and are followed later by tumours. The interval between the appearance of the pigment and that of the tumours may be of many years duration, as Parkes Weber has pointed out. Many cases when first seen are incomplete and

lack either the tumours on the nerves or the superficial tumours.

**TREATMENT** There is no treatment except surgical removal of any tumours which by their size or position are causing inconvenience.



FIG. 9.  
von Recklinghausen & Dineen.



Adenomatous naevi (except sebaceous cysts) are rare and cannot be considered here.

**Adenoma Sebaceum** (Naevus Pringle of Continental dermatologists). Small red or yellowish tumours about 2 mm. in diameter arranged symmetrically on the face, especially near the sides of the nose, and appearing usually in early childhood. They are of congenital origin and differ in histology and consequently in appearance in different types. *Pringle Type*. Hyperplasia of sebaceous glands, hair follicles and blood vessels, hence the tumours are red. This is the commonest form. *Balcer Type*. Hyperplasia of sebaceous glands, hair follicles and sometimes sweat glands, but not of blood vessels, so the tumours are yellowish. *Hallopeau Leredde Type*. Hyperplasia of fibrous tissue with some hyperkeratosis, hence the tumours are brownish and warty.

The tumours are usually first noticed in childhood from three to five years of age and may increase in number towards puberty after which new lesions cease to appear though the old ones remain. The condition is met with in normal children though it is much commoner in mentally defective children and epileptics. In these it forms part of a syndrome called Epiloia which includes also tubercle sclerosis of the brain (causing epilepsy paralysis and idiocy or feeble-mindedness) vascular naevi, pigmented patches and soft neurofibromata of the skin resembling those of von Recklinghausen's disease (p. 68) and sometimes also fibromata of the kidneys, heart, and other abdominal organs. Any part of the syndrome may occur by itself and adenoma sebaceum frequently does so. It may occur sporadically or may be hereditary and familial.

**TREATMENT** The little tumours on the face may be destroyed by electrolysis or galvano-cautery (p. 41).

**Sebaceous cysts Atheromata Wens.** These are usually regarded as retention cysts of the sebaceous glands, but it is probable that they should be looked upon rather as forms of sebaceous gland naevi. They are found most commonly on the scalp, scrotum and vulva, and seldom appear before puberty a point of distinction from dermoid cysts. They may be of any size up

to an inch or so in diameter are usually yellowish or white in colour and often have a dimple at one point representing the opening of the sebaceous gland

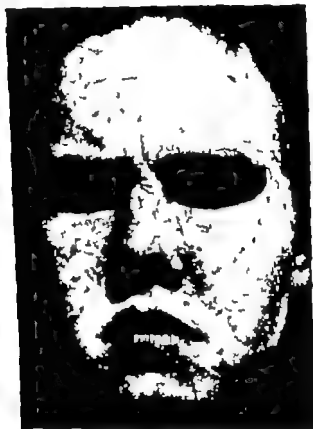


FIG. 10.

Adenoma Sebaceum. Pringle Type

HISTOLOGICALLY they consist of a wall of epithelium enclosing a mass of cheesy or semi-fluid sebaceous and horny material. Outside the epithelial wall is an adventitious capsule of compressed connective tissue

TREATMENT Excision, including the epithelial wall. If the wall is left the cyst will probably reform.

## CHAPTER V

### SKIN AFFECTIONS DUE TO PHYSICAL CAUSES

**Pressure**—Continuous pressure on the skin causes atrophy *e.g.* under the pad of a truss. Intermittent pressure especially if combined with friction, causes hypertrophy of the horny layer as in the callosities on the hands of manual workers—a protective response. When the thickened horny layer is unable to project above the surface owing to external pressure it projects inwards and causes a corn. A corn consists entirely of a mass of horn cells forming a cone with its apex directed inwards. This presses on the nerve endings in the corium beneath and causes pain. A corn is seldom formed except where a bony prominence underneath concentrates the pressure on to a small area of skin.

**TREATMENT** Frequent paring at intervals of a few days combined with the removal of pressure by wearing a ring of adhesive felt around the corn allows the cone-shaped plug to be gradually extruded. A 400r dose of X rays often reduces the pain of a corn and also its rate of growth, and enables it to be removed more quickly. In a very persistent corn the bony prominence underneath must be removed if cure is to be permanent.

**Moisture** Prolonged wetness of the skin causes maceration of the horny layer. This then ceases to form an efficient protective layer and is more liable to injury and to infection. The macerating effect of water is greatly increased if alkali is present, *e.g.* in washerwomen, charwomen etc. for the alkali dissolves out the protective sebum. When such skin does dry it is rough, red and cracks easily. Another result of constant wetness is seen in cooks, barmaids etc. This is an infection of the nail-folds, with monilia or other organisms, which causes chronic swelling and redness of the nail-fold (paronychia p 155)

**Dryness.** In a dry atmosphere the horny layer tends to crack owing to loss of elasticity. This is seen in a minor degree during periods of east wind in this country. The remedy is the application of grease which softens the horny layer and also helps it to retain its moisture.

### SKIN AFFECTIONS DUE TO COLD

Moderate degrees of cold (0-5° C. 32-41° F.) cause a contraction of the minute vessels of the skin as well as of the strong arterioles and the larger arteries, resulting in a white cold skin. The minute vessels soon dilate but the arterioles and arteries remain contracted for a long time. The skin is then red though still cold because the metabolism of the tissues is so reduced by the cold that oxygen consumption virtually ceases and the haemoglobin is no longer reduced. A very slight degree of cold (15° C. 59° F.) is able to keep the arteries contracted once they have been caused to contract by a somewhat lower temperature. At temperatures of about 10-15° C. (50-59° F.) the cold red skin often shows some degree of cyanosis, but a conspicuous cyanosis in response to cold is an abnormal reaction (Lewis). Prolonged exposure to cold leads to a paralytic distension of the minute vessels, some of which may become visible to the naked eye. Intense cooling of the skin leads to freezing at a temperature of -6° C. (21° F.). This is followed on thawing by a necrosis of the frozen cells including those of the blood vessels, leading to thrombosis and necrosis of the frozen tissue and constitutes frost bite. Frost-bite is most apt to attack points such as the fingers and toes, ears and tip of the nose. It may occur even at temperatures just above freezing point when the skin is wet and is being cooled by a strong wind. The effects of frost-bite are mainly due to thrombosis of the vessels and differ in degree according to the completeness with which this takes place.

**CLINICAL FEATURES.** The affected skin becomes numb and white. On thawing it becomes painful, bluish red, and oedematous. Bullae appear and sloughs are formed which eventually separate. The sloughs may represent only the superficial parts of the skin or may include whole digits.

**TREATMENT** The affected part should be kept as completely at rest as circumstances permit, it should be thoroughly but most gently cleaned and wrapped in dry sterile dressings and many layers of wool. The patient should be given extra clothes and hot drinks but no alcohol. Thawing must be done slowly and early Heparinization should be used to limit the extent of the thrombosis. Massage and artificial warmth to the part are absolutely contraindicated. If at a great height the administration of oxygen is extremely helpful. Antitetanus serum should be given. If sepsis is avoided there should be no hurry to resort to surgery. (Raymond Greene *Lancet*, 1940 1 303)

**Immersion Foot.** A condition brought on by immersion of the legs and feet for hours or days in cold sea water as occurs in waterlogged boats or rubber dinghies. The feet are at first for some hours or days cold swollen, discoloured and numb. The peripheral arteries may be impalpable and gangrene may occur. The hyperaemic stage follows after rescue and lasts six to ten weeks. The feet are swollen red hot and intensely painful, sensory changes and motor weakness occur blisters, ulcers, and gangrene may develop nails may be lost. The post hyperaemic stage lasts weeks or months. There is a cold hyper or hypohidrotic foot with all the manifestations of a chronic sensory neuritis.

**TREATMENT** The essence of successful treatment of the hyperaemic stage is to be extremely gentle and to keep the feet cool and aseptic. The rest of the body should be kept warm but the feet should be exposed to cool air and if necessary cooled by an electric fan or icebags. Open sores, blisters, and areas of gangrene should be kept dry. (M Critchley *Med Ann* 1943 p 175)

**Perniosis (Haxthausen)** This is a name which embraces ordinary chilblains, acrocyanosis, erythrocyanosis crurum, certain types of arborescent dilated venules on the legs and livedo reticularis. All these are due to the effects of persistent mild degrees of cold acting on a person whose cutaneous vessels are peculiarly susceptible. The essential histology is the same in all of them (Haxthausen) and only differs in degree.

It consists in a dilatation of the minute vessels and small veins and a constriction of the arterioles. This constriction can be relaxed by considerable prolonged warmth but is very rapidly re-established on exposure to a mild degree of cold hence some people may have chilblains even in a cold summer. The dilatation of the capillaries and veins is very persistent and in well marked cases lasts throughout the year. In pronounced cases collections of leucocytes forming hyaline masses are found within the vessels. There is also a proliferation of the connective tissue of the corium and a perivascular round-cell infiltration.

**Chilblains** (*Erythema pernio*). These are dusky red round or oval, intensely itchy swellings of the skin usually on the fingers or toes, which occur in susceptible people in cold damp weather. They are of variable size and when acute are often vesicular. Occasionally they ulcerate. They are most common in young female adults. Diet inadequate in protein and fat, anaemia and tuberculosis are predisposing causes.

**DIAGNOSIS.** Lesions which may be mistaken for chilblains are papulo-necrotic tubercles (p. 216) and lupus erythematosus (p. 303). The former are smaller and more regular in size than chilblains they occur also on the elbows, shoulders, and legs, and leave small white depressed scars. Ordinarily chilblains leave no scars but if they become ulcerated they leave irregular scars. Papulo-necrotic tubercles are apt to occur in people who have acrocyanosis and possibly actual chilblains as well. Lupus erythematosus when it occurs on the hands usually forms oval plaques or rings on the backs of the fingers these are bluish red and may be scaly or may show the characteristic stippling. Other lesions of lupus erythematosus will most likely be present also on the face.

**TREATMENT.** Warm clothing is essential, and this applies as much to the trunk as to the extremities. On the whole, vasodilator drugs have proved disappointing but some improvement may be obtained from nicotinic acid 25-50 mg. four times a day, tolazoline hydrochloride (Priscol) 25-50 mg. thrice daily or dibenylene 10-20 mg. thrice daily. The latter is probably the most effective but the side-effects may be troublesome. When the

chilblains have developed, painting with tincture of iodine or ichthyol, rubbing with ointments containing menthol, methyl salicylate or iodine, or the application of lead lotion are useful.

Severe chilblains can be incapacitating and then sympathectomy may be indicated. Of physical methods galvanic hand or foot baths (the bath being made the negative pole and the positive applied as a pad higher up the limb) diathermy and ultra violet light, both general and local are the most useful. X-rays may be used to remove the thickening caused by a persistent chilblain.

Acrocyanosis is the name given to a persistent blueness and coldness of the hands on exposure to cold. It may be associated with some hyperidrosis of the hands and feet. The treatment is on the same lines as for chilblains and sympathectomy is effective if the symptoms warrant it.

Erythrocyanosis crurum is a condition most common on the outer side of the lower third of the leg in young women. It has become much more common of recent years since skirts became short and stockings thin or absent. There is a cold bluish red swelling over an area 3-4 inches in diameter on each leg. On the surface there may be visible a number of dilated venules forming a pattern like the tributaries of a river on a map and chilblains, sometimes ulcerative, are occasionally superimposed, producing the perimorphic type of erythema induratum (p 215). Once established the condition is extremely hard to get rid of. In the summer the affected area is hot, swollen and red. According to Boyd it is due to a congenital inadequacy (small size) not only of the arteries of the leg but of the aorta. Vasodilator drugs ultra violet light or lumbar sympathectomy give some relief but the best treatment, especially if aching is complained of or chilblains are present is the wearing of elastic stockings. Sympathectomy will relieve the effect of cold but has no effect on the swelling which occurs in hot weather.

Livedo reticularis (marbled skin) This is a bluish red net work enclosing islands of whiter skin seen particularly in children and adolescents, usually on the legs but sometimes also on the arms. It is most noticeable when the skin is cold. It appears to be produced as a result of the way in which the

PLATE I



Erythrocytic Crinum





arterial blood is delivered to the skin. The white islands are the areas which have a relatively direct supply from below the bluish network representing the intervening areas whose arterial supply is mainly through anastomoses. (Adamson) The effect of cold in causing spasm of the arterioles and dilatation of the minute vessels is naturally shown first in those areas which have the less direct supply. This network in which the circulation is slower is the seat of the first appearance of the measles rash (Adamson, Lewis), of certain drug eruptions and sometimes of lichen planus and parapsoriasis (Fox, MacLeod) According to Adamson the roseola of syphilis occurs at the "knots" in the network. When heat is applied frequently to the skin a heavy deposit of melanin is formed in the basal layer of the epidermis over the bluish network, so that a dark brown network is formed, the so-called *Ephelis ab igne* (Fig 11). It is commonly produced on the legs by heat from a fire and on the abdomen by that from hot-water bottles, e.g. in cases of gall stone.

Raynaud's Disease is produced in a susceptible individual by a suitable degree of cold (about  $17^{\circ}\text{C}$   $63^{\circ}\text{F}$ ) causing a spasm of the digital arteries. (Lewis.) As it is dealt with in text-books of medicine it will not be further considered here.

### SKIN AFFECTIONS DUE TO HEAT

Exposure to heat causes a dilatation of all the vessels of the skin, arterioles as well as minute vessels and venules. The result is an immediate erythema which, however soon disappears when the source of heat is removed. Excessive heat causes the various degrees of burns (dry heat) and scalds (moist heat). These are dealt with in text-books of surgery. The pigmentation due to heat (*Ephelis ab igne*) has been considered above.

Electric burns are caused by the heat evolved in the passage of a heavy electric current through the skin, complicated by the electrolytic effects of the current. They are generally very slow in healing. Death in electric shock is usually due to paralysis of the respiratory centre, and many such cases revive if artificial respiration is begun at the earliest possible moment and continued if necessary for not less than two hours.



*For Archibald Gray's Case.*

FIG 11.

*Ephelis ligna.*

## SKIN AFFECTIONS DUE TO RADIANT ENERGY

(*Sunlight, Ultra violet light X rays Radium rays*)

**Sunlight.** The effects of sunlight on the skin are best divided into those which are produced in normal individuals and those which only appear in people who are, in one way or another especially sensitive either to the visible or to the ultra violet rays of the sun.

**Acute solar dermatitis.** In normal individuals exposure of the skin to strong sunlight, e.g. in the early summer before protective pigmentation has developed is followed in about six hours by itching, burning, redness and some swelling proceeding possibly to actual vesicle formation. If the face is affected the eyes may be closed by oedema and there may be in addition conjunctivitis and damage to the cornea ( snow blindness ) due to the action of the ultra violet rays. Severe degrees of acute solar dermatitis are most likely to occur after exposure to strong light on ice, snow water or sand because of the strong reflection of ultra violet light from such surfaces. The affected epidermis eventually peels off and new and thicker horny and prickly-cell layers are formed together with a development of pigment (melanin) in the basal layer.

A severe attack may take a week to subside, milder cases only a day or two.

**DIAGNOSIS.** Usually easy but cases are sometimes mistaken for erysipelas. In acute solar dermatitis, however the temperature is not, or is only very slightly raised, and the eruption is bilateral and symmetrical.

**PREVENTION.** The application, before exposure, of red or brown grease paint, calamine lotion, 10% tannic acid in 25% spirit, or yellow petroleum jelly with or without 2% quinine or 10% salol.

According to H. G. Harry menthyl salicylate 10% in an ointment base is extremely effective even against a subtropical sun. Six per cent of salol in an emulsion of equal parts of linseed oil and lime water is suggested by Goodman. Recently para-aminobenzoic acid 15% in vanishing cream or 10% in 70% alcohol has been advised as a reliable protective. Esters of para

## 80 SKIN AFFECTIONS DUE TO PHYSICAL CAUSES

aminobenzene acid should not be used as they are more liable to sensitise the skin, although they are more convenient pharmacologically (8 Rothman)

**TREATMENT** Evaporating lotions such as Lotion plumbi and Lotion calaminæ and later cold cream or zinc cream. Hydrocortisone lotion is particularly effective.

Pigmentation is a natural result of exposure to sunlight in normal individuals and it develops without any antecedent acute dermatitis, provided that the exposures are not too intense at first. Contact with tar greatly increases solar pigmentation.

Freckles are small brown pigmented macules, which occur in fair-skinned and especially in sandy or red haired individuals as a result of exposure to light. It appears as though the pigment forming capacity of such skins were only present in spots, viz where the freckles develop

**PREVENTION** Avoidance of exposure to strong light or the application before exposure of agents such as those described above for preventing acute solar dermatitis.

**TREATMENT** Application of  $H_2O_2$  10 vols., after lightly scraping the epidermis over the freckle, or the application to each freckle of pure carbolic acid on a pointed match stick. The acid should be wiped off with methylated spirit as soon as the skin has turned white. Peeling of the freckled skin may be tried by the frequent application of 1% hydrarg perchlor in 50% spirit.

**Chronic solar dermatitis** Normal individuals, especially if fair-skinned who have been exposed to strong sunlight for many years develop a condition called chronic solar dermatitis or sailor's skin. It is characterised by atrophy wrinkling pigmentation (more intense in patches) white atrophic patches, telangiectases, and warty growths (solar keratoses) some of which eventually become the seat of basal or squamous-celled epitheliomata. It is most commonly seen on the face neck and backs of the hands of those whose work has kept them in the open air especially in sunny countries or at sea.

**TREATMENT** No treatment will restore the original condition of the skin but ointments may be used to soften it and the



FIG. 12.  
Summer Eruption.

## 82 SKIN AFFECTIONS DUE TO PHYSICAL CAUSES

keratoses can be removed by salicylic acid ointment, salicylic collodion, freezing with CO<sub>2</sub> snow scraping excision, radium, or X rays

**Peasant's Neck** (Rasch) (*Cutis rhomboidalis nuchae*) is a form of chronic solar dermatitis in which the skin of the back of the neck is thick and red and divided by creases into rhomboidal areas.



FIG 12

Summer Eruption. Characteristic distribution on face.

In individuals who are abnormally sensitive to sunlight various conditions may develop as a result of exposure to it. One of the commonest is the Summer Eruption of children (Hutchinson's Summer Prurigo Rasch's "Polymorphic Light Eruption") This is a polymorphic eruption of erythema papules, vesicles weeping areas,



FIG. 14.

Sir H. Graham-Litch's Case.

*Hydroa aestivum.*

crusts pigmented macules and small depressed scars which occurs on the exposed parts (face neck, forearms, hands and knees) in children (Figs. 12 and 13). It usually appears in spring or early summer lasts a few months and then disappears to recur again in the succeeding summer. The parts of the spectrum which produce this eruption are the orange, yellow and green (Eklidnow *Brit. Jour. Derm. & Syph.*, 1935 47 27"). In some cases the eczematous condition which develops lasts throughout the winter. Owing to the scratching secondary infection and lichenification (p. 287) may occur.

**ETIOLOGY.** Obscure. There is some evidence that the breakdown products of the epidermic cells are responsible (Goldsmith). No sensitising substance can be found in the urine, faeces or blood serum. Most of the patients grow out of their sensitiveness about puberty but in some it persists into adult life.



## 86 SKIN AFFECTIONS DUE TO PHYSICAL CAUSES

**TREATMENT** Mild degrees of X ray dermatitis are best treated with hydrocortisone ointment though simple applications such as lead or calamine lotion or zinc cream are also effective. If bullae form, they should be opened and kept aseptic by combining the hydrocortisone with an antibiotic e.g. 1% Neocortef ointment.

Severe degrees of radio-dermatitis with ulceration may never heal. The only treatment for these is total removal of the damaged tissue either by severe scraping, or by excision followed by plastic repair.



FIG. 16.

Chronic X Ray Dermatitis under Left Jaw Ulcer unhealed for 25 years following X Ray treatment of tuberculous glands

Small areas of ulceration appearing after some years may sometimes be got to heal by application of an antibiotic, such as neomycin or tetracycline and these sometimes prove more effective if combined with hydrocortisone. Sometimes they may be healed by zinc ionisation but they often require excision and grafting.

Chronic X ray dermatitis is the result of frequent exposure to small doses of X rays over a prolonged period such as may occur accidentally in X ray workers. It usually affects the



FIG. 16.

Damage to Finger Nails from handling Radium.



FIG. 17

Malignant ulceration due to handling Radium (F tal).

## 88 SKIN AFFECTIONS DUE TO PHYSICAL CAUSES

backs of the hands and fingers. The affected skin is dry hairless and atrophic, and tends to crack easily. The surface shows patchy pigmentation and telangiectases. Itching and burning are felt especially in cold weather. Later on small warty keratoses appear. These may be knocked or rubbed off and leave superficial ulcers, or they may increase in size and become squamous epitheliomata. The atrophic skin is easily infected by pyogenic organisms. The nails become opaque, uneven, brittle and split easily and infection of the surrounding tissues is apt to occur. The subcutaneous tissues and fat atrophy and the bones may become softened.

**TREATMENT** The treatment is only palliative. The hands should be protected and the condition treated much as eczema (p. 331). Soap and water should be avoided and the hands cleaned with olive oil or cold cream. Warty growths should be removed by excision, diathermy or CO<sub>2</sub> snow.

**Radium dermatitis** This is very similar to that produced by X rays, only it is usually smaller in extent. It should be treated on the same lines.

## CHAPTER VI

### DERMATITIS DUE TO CHEMICAL CAUSES (DERMATITIS VENENATA)

DERMATITIS is inflammation of the skin, and it is characterised therefore by redness, swelling heat and pain, or more often by itching rather than pain. The appearances of dermatitis vary according to its severity and the stage of its evolution. The earliest sign is erythema or redness this is followed or accompanied by some swelling of the skin due to oedema. Vesicles or bullae may then appear if these rupture or get rubbed off, their bases exude serum (weeping dermatitis). Later the serum dries up to form crusts. The process may go on to resolution, the horny layer re-forming under the crusts or relapse with further weeping may occur. Owing to the oedema of the prickle-cells the granular and horny layers are imperfectly formed, the horn cells being nucleated and sticky and adhering together to form visible scales (scaly dermatitis). The picture may be complicated by the effects of scratching and the inoculation of pus organisms, streptococci and staphylococci.

**HISTOLOGY** The blood vessels of the corium are dilated and there is an exudation of lymph and an infiltration of small round cells, chiefly around the vessels. In the epidermis the cells are swollen by imbibition of fluid (spongiosis) and separated by the collection of fluid between them i.e. there is an inter and an intra-cellular oedema. Here and there the "prickles" give way under the tension and spaces filled with serous fluid are left between the cells. These increase until they form visible vesicles.

The chemical substances giving rise to dermatitis may reach the skin from outside or via the blood stream. In the case of substances reaching the skin from the outside they may be of

## 90     DERMATITIS DUE TO CHEMICAL CAUSES

so irritating a nature as to produce dermatitis in any normal person *e.g.* mustard-gas, croton oil, (traumatic dermatitis), or their irritant properties may only affect certain individuals, *e.g.* paraffin detergents. On the other hand, they may be of



FIG 18.

Traumatic Dermatitis. Acute    eczema

such a nature as to produce no effect on normal individuals but cause a dermatitis by an allergic mechanism only in persons who have become sensitised to them by previous exposure, *e.g.* primula, rhus picric acid etc (sensitisation dermatitis). Cases of dermatitis due to irritants conveyed by the blood stream are usually examples of sensitisation dermatitis.

### ANIMAL IRRITANTS

“Woolly bear caterpillars” Contact with the poison hairs of these caterpillars may set up an irritating urticarial rash. Wet dressings of lead and calamine lotion with the addition of 3% phenol to allay the irritation are usually effective.

*Jelly fish.* A severe sting by the stinging cells on the tentacles of a jelly fish can be very disabling. The sting feels “like a red-

hot wire on the skin" and may soon be followed by shock abdominal rigidity feeling of constriction of the chest, difficulty in respiration and, in children, even unconsciousness. The local results are cord like raised wheals on a swollen red base and an intense burning pain. The application of sat. ammon. aromat. is said (J Fawcitt) to abolish the local pain. Morphine may be required for the more general pain and distress. The vomiting and constipation which follow in some cases may be due in part to the morphine. The symptoms may take from 2 to 3 days to pass off. In mild cases the application of lead and calamine lotion with the addition of 2% carbolic acid to allay the irritation may be sufficient (see H. Muir Evans, *BMJ* 1945 2 165).

*Weever fish and sting rays* The poisonous spines of these fish can inflict very painful wounds with general symptoms similar to those of severe jelly fish stings (see above). The injection of a few minims of 5% solution of potassium permanganate down the track of the spine has been recommended as well as morphine for the general symptoms.

*Insect bites and stings* see p 148.

## VEGETABLE IRRITANTS

About one hundred different plants are known to be capable of causing a dermatitis in susceptible people. The commonest in this country are *Primula obconica*, *Primula sinensis*, May weed (*Anthemis cotula*) Wild Parsnip Tulips, *Chrysanthemums*, Tomato plants, Celery Narcissus and Daffodils. The common Ivy (*Hedera helix*) occasionally causes a dermatitis. Some members of the *Rhus* family notably *Rhus toxicodendron* (Poison Ivy) are very poisonous but not often encountered in this country. Chinese and Japanese lacquer derived from the latex of *Rhus vernicifera* causes a dermatitis in some people. Vanilla pods sometimes cause a dermatitis in those who handle them while mustard, turpentine croton oil, and arnica are used for their irritant properties which sometimes get out of hand. Celery carrots and other vegetables may cause dermatitis in those employed in canning them. Certain woods—for example, teak, East Indian satinwood, ebony and Cocos wood—used for flutes, are

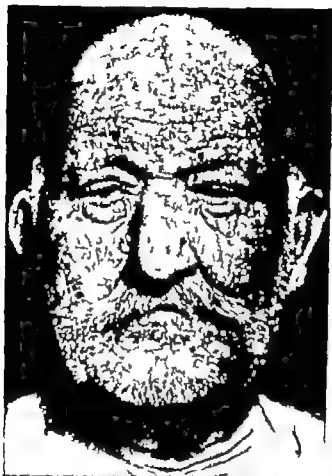


FIG. 19

Chronic Separation Dermatitis. Apparently due to H<sub>2</sub>S

irritating to some of those who work with them. O'Donovan has recorded the dermatitis produced by Podophyllum resin in those who grind it. Dore and Prosser Thomas have described nine cases of dermatitis in workers in a morphine factory (*Brit Jour Derm. & Syph* 1911 58 177).

R. Klaber (*Brit Jour Derm. & Syph.*, 1913 54 193) called attention to the fact that a number of plants cause a dermatitis



FIG. 20

One leaf of *Rhus Toxicodendron* "Poison Ivy" (left), with two leaves of a harmless *Virginia creeper* (right) for comparison.

only when the skin has been exposed to sunlight after contact with the plant or its juice, a condition which he calls phyto-photo-dermatitis. Such plants are parsnips, both wild and cultivated cow parsnip (*Hieracium*) some other Umbelliferae and some Rutaceae (see lime bergamot) also figs (*Moraceae*) and agrimony (*Rosaceae*). The rays concerned lie in the blue-violet and longer ultra violet regions.

An example of phyto-photo-dermatitis is an eruption which sometimes occurs in people who after bathing on a sunny day lie on the grass without interposing a towel. Between 24 and 48 hours later an eruption appears on the areas of skin which have been in close contact with the vegetation and later exposed to the sun. It consists of linear bullae on a red base. These lie parallel to or cross one another in a bizarre fashion. The bullae disappear in a few days but pigmentation remains at their sites for weeks.





FIG. 21

Hand showing dermatitis.  
*Dermatitis bullosa striata*  
*prutenica* (Oppenheims)

or months. This eruption was first described by Oppenheim in 1906 in Vienna and is known as *Dermatitis bullosa striata pratensis* or more shortly as *Meadow Dermatitis*. A number of plants appear to be capable of producing it among them wild parsnip (*Pastinaca sativa*). Lead and calamine lotion is usually the only application required.

**CLINICAL FEATURES OF PLANT DERMATITIS.** The onset is usually acute and begins an hour or two after contact. The hands, face and (male) genitals are covered with closely set minute vesicles on an erythematous base. The eyes may be closed by oedema of the lids and the scrotum may be much swollen. The irritation is usually intense. On the face, plant dermatitis is often mistaken for erysipelas, but the sudden onset, affection of both sides simultaneously absence or slightness of fever and history or probability of contact with an irritant plant, should help to distinguish the two conditions. A patch test is often helpful. (Fig 29, see also page 330) Many plant dermatitis are due to sensitisation and do not therefore occur at the first contact, and may do so only after the patient has handled the plant many times with impunity.

**TREATMENT** An attack may sometimes be prevented even after exposure by very thorough washing of the face and hands with ordinary household soap repeated several times, to remove the irritant, which is usually of an oily nature. J B Howell (*Arch. Derm. & Syph.* 1913 48 373) has found experimentally that no measures prevent dermatitis from developing in a sensitive person after contact with poison ivy except the application of 5% solution of potassium permanganate within fifteen minutes of exposure. When the attack has developed cold wet dressings of potassium permanganate 1:5000 to 1:3000 are recommended (Andrews). Later on, or in slighter cases, lead and calamine lotion with or without 1% or 2% of phenol, may be used and finally zinc cream. Hydrocortisone lotion or ointment are also useful in any stage. The patient must be careful to avoid contact with the offending plant in the future as subsequent attacks may be more severe. Palmer in Cornwall has had some success in desensitisation against "Lily Rash" due to

Narcissi and Daffodils by the use of a vaccine made from the leaves and stalks of Daffodils (Palmer W H. *Lancet* 1834 2, 755)

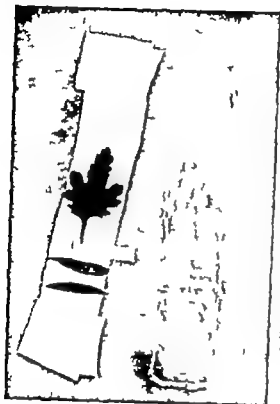


FIG. 22.

Positive Patch Test in case of suspected *Chrysanthemum* Dermatitis.

The application of the leaf and petals to the skin of the middle of the back under strapping for 24 hours caused erythema and vesication over areas exactly corresponding to the shape of the leaf and petals. This illustrates the fact that the whole skin usually becomes sensitized, not only that portion of it which is habitually in contact with the antigen.

## MINERAL IRRITANTS INDUSTRIAL DERMATITIS.

Dermatitis caused by external contact with mineral irritants forms a large and increasing proportion of the skin diseases met with in practice for it includes most of the cases of industrial and occupational dermatitis, of which new causes are arising almost daily as new manufacturing processes are introduced. The subject has very great practical importance because of the claims for compensation which arise.

It is therefore essential that a practitioner seeing a

case which may be one of industrial dermatitis, should make careful notes, for these may be valuable later in case of a claim for compensation. The majority of cases of industrial dermatitis arise on the hands or forearms which are actually in contact



FIG. 22.

PERIO ACID DERMATITIS, left wrist.

Note large superficial vesicles of varying size and contrast. Also the small, uniform, pin-head sized vesicles of eczema. Figs. 141, 142, 143, 145

with the irritant. Where the trouble is due to a liquid which splashes, or to a spray the face may also be affected, while in cases due to dust the neck, the axillae, the perineum and genitals, and the feet and ankles may be involved owing to the dust accumulating in the clothing and getting rubbed into the skin at these places. Moreover if a patient has for some time had a severe dermatitis from any cause on one area it may suddenly spread to other areas not previously involved (vide eczema, p. 318). If a particular eruption always improves during week-ends or holidays when the patient is away from work, or if it came on shortly after his beginning a new job or

if fellow workmen are similarly afflicted, the probability of its being due to his work is increased. On the other hand, the fact that a man has worked for years at a particular job without getting dermatitis is no evidence that his eruption is not due to his work for some modification may have been introduced into the substance he handles or he may at last have become

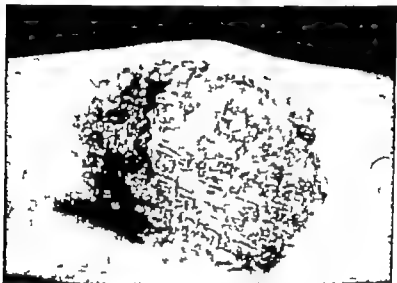


FIG. 24

*Ichthyos Dermatitis, arm.* Due to painting *tinea arcuata* with Thact. Iodl.  
See note to Fig. 23 (contrast with eczema).

sensitised to some chemical substance he encounters daily. Another factor to be borne in mind is that many cases of industrial dermatitis are due not to the substances with which the patient works but to those with which he cleans his skin after work, e.g. soda paraffin, coarse sacking. It must also be remembered that many cases of alleged industrial dermatitis have nothing to do with the patient's occupation at all but are ordinary cases of constitutional eczema, scabies, psoriasis, lichen planus, etc.

After a patient has been off work for some time on account

of industrial dermatitis, psychological factors begin to play a larger and larger part in the continuance of the eruption which is not likely to clear up until the question of compensation is finally settled.

Mineral irritants may be classified as follows (after Sequeira)

- 1 Those which cause mechanical injury *e.g.* inert dusts, sand, pumice, silicates.



FIG. 23.

Chemical Dermatitis from Iodine in an Iodine Worker

Those which macerate the horny layer *e.g.* water and alkalis (soap, soda, lime and ammonia)

- 3 Those which dissolve out the fat, *e.g.* petrol, paraffin, benzine, turpentine and its substitutes.

- 4 Strong irritants, *e.g.* chromic acid and its compounds, coal tar derivatives, aniline compounds, dyes, phosphorus, arsenic, strong acids, and caustic alkalis.

5. Mineral oils, as used for lubrication.

In the U. S. A. it has been estimated that the ten commonest causes of industrial dermatitis are

- 1 Petroleum oils and greases. 19%.

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- 2 Alkalis, including cement and concrete. 12%.
- 3 Solvents. 8%
- 4 Plants and woods. 6.5%
- 5 Metals and metal plating 6%
- 6 Rubber and its compounds. 3%
- 7 Chemicals (unspecified) 3%
- 8 Paints, Enamels and Varnishes. 3%
- 9 Acids and acid fumes. 2%
- 10 Dyes and dye intermediates. 3%



FIG. 26

Ulceration due to chromic compounds. "Chromic Holes."

**TYPES OF DERMATITIS PRODUCED BY MINERAL IRRITANTS.** All the stages referred to on page 80 may be seen with in addition hyperkeratoses and fissures of the palms, suppurative folliculitis produced by the irritant entering the skin at the pilo-sebaceous follicles, ulceration of the hands and nasal septum (chromic compounds) and finally squamous epithelioma (due to tar and its products, paraffins soot arsenic and mineral oils) Squamous

epitheliomata due to mineral irritants are usually found on the hands, forearms, face and scrotum. A long period of exposure is usually required, 10-20 years,<sup>1</sup> and the growths may not appear for another 10-20 years, perhaps long after the patient has given up the work in question. The finger nails are often affected in chemical dermatitis, they may be stained, eroded brittle or separated from their beds.

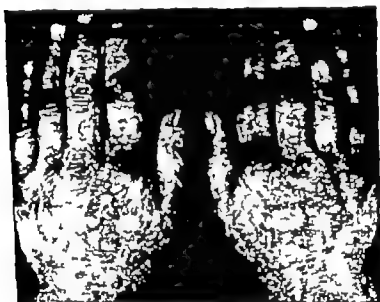


FIG 27

Dermatitis in photographic emulsion maker ? due to formalin.

It is impossible in a book of this size to treat the subject of industrial dermatitis in any detail. It must suffice to indicate a few of the occupations in which industrial dermatitis most commonly arises. Housewives, charwomen, washerwomen, cooks and those who wash up dishes in hotels and restaurants commonly suffer from an *alkali dermatitis* of the hands due to water soap, and soda. For *detergent dermatitis* see page 103.

Much shorter periods have been recorded, especially in the case of burns from hot tar.



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Cooks and dishwashers also commonly suffer from chronic infections of the nail fold with *monilia* and other organisms (p 185) Bakers sometimes suffer from *dermatitis* of the hands and forearms. This is not due to any one cause but is produced in some by *ammonium persulphate* added to the flour as an



FIG. 23

Dermatitis medicamentosa. Result of ointment  
 applied around an ulcer on peroneal leg  
 margin and large bullae. Note sharp

“improver” in others by salt sugar or flour or in others again by rubbing the half-dried dough off the forearms with coarse sacking. Builders are apt to get dermatitis of the exposed parts, and of the feet and ankles from lime and cement. Chimney sweeps often get a dermatitis of the *scrotum* due to tarry material in the soot giving rise eventually to *emithelioma* (chimney

sweep a cancer) Workers in tar and its products often develop tar acne and generally suffer in time from a chronic dermatitis of the exposed parts leading to *warty growths* and eventually to *epitheliomata*. The same applies to workers in the Scottish shale-oil industry. Those who attend spinning mules leaning over to tie broken threads get the front of their trousers wet with lubricating oil and many of them subsequently develop *symptomatic epithelioma of the scrotum* (mule spinner's cancer). Metal turners and lathe operators often get oil acne, i.e. comedos and follicular papules and pustules on areas which are habitually splashed with lubricating and cutting oils e.g., fore-arms, face and sometimes legs. Heavy oils tend to cause oil acne and light oils oil dermatitis.

In aeroplane factories (Schwartz and Russell 1941 *Pub Health Reports* Washington, 56 581) dermatitis may arise from the protective grease or varnish used to cover sheets of duralumin and other metal alloys. The anodising process may result in dermatitis from chromic nitric or hydrofluoric acids. De-greasing of the skin by trichloroethylene and dope solvents may cause dermatitis if the fat is not replaced. In the paint shops solvents (thinners drying oils resins and the pigment sac chromates may all cause dermatitis. In welding, metal fluxes containing fluorides may cause nasal irritation, ulceration and epistaxis.

In shell-filling factories dermatitis may be caused by the explosives themselves or less often by the solvents used.

*Synthetic waxes* (chlorinated naphthalenes) used for the electrical insulation of wire and in condenser manufacture often set up a chloracne (p 401) after some months of exposure. This appears first on parts exposed to the vapour from the wax and later on covered parts.

Nickel platers all eventually develop a dermatitis due to nickel salts. Chromium platers are apt to develop chrome dermatitis and ulcers on the hands and on the nasal septum. Makers and users of phenol formaldehyde and urea formaldehyde resins and glues sometimes develop a dermatitis from the formaldehyde. Photographers sometimes get a dermatitis of the fingers due to metal and alkalis. French polishers commonly

suffer from dermatitis of the hands due to *bichromates* and *turpentine*. Surgeons and nurses sometimes develop a dermatitis of the hands due to *antibiotics* (*penicillin*, *streptomycin*) *chlorpromazine* and *antiseptics* dentists one due to *procaine* pathologists one due to *formalin*. The latter is very prone to recur on the slightest contact with the drug

**Household Detergents** (See ■ *Hodgson Practitioner* 1953 170 166) The modern blended synthetic detergents cause



FIG. 29.  
Dermatitis due to "anti-sweating" lotion.

dermatitis of the hands in a certain number of those who use them. These detergents are of very various composition but most of them contain as much alkaline phosphate as detergent. The way in which detergents act in irritating the skin is not fully understood but defatting and drying of the horny layer occurs as with alkaline soaps. Apparently there is also some damage to the keratin molecules with liberation of sulphhydryl groups (E. J. Van Scott and J. B. Lyon *J Invest Derm.*, 1953, 21 199) An acute contact dermatitis, with swelling redness, blistering weeping and crusting may occur or a dermatitis resembling eczema may develop more slowly and be accompanied by brittleness and breaking of the nails and fissures on the knuckles. Paronychia inflammation may also occur (See page 155.) Dermatitis may occur on areas of skin in contact with cloth-

ing which has been washed in detergents without thorough rinsing

**PREVENTION** Detergents should always be used in the minimum concentration necessary for the work in hand and should always be well rinsed out of clothing washed in them. After wards, the hands and the forearms should be well rinsed in clean water and a cold cream or similar greasy preparation should be rubbed in. Those whose skins are liable to irritation by these substances should wear rubber gloves, preferably those lined with "suedette" or plastic mittens.

**TREATMENT** of detergent dermatitis is on the usual lines for eczema and dermatitis. (See page 330)

**PROPHYLAXIS OF INDUSTRIAL DERMATITIS** This includes:— Selection of suitable personnel those with thick, dark, oily skins are as a rule less susceptible than those with fair thin, dry skins those with relatively hairless skins are less liable to folliculitis from oils and greases than those with very hairy arms and legs. Cool conditions of working, because heat and perspiration tend to increase risk of dermatitis. Cleanliness of both works, workers, and workers clothes. Prevention of all unnecessary exposure to irritants. Mechanical protection by gloves, aprons, etc. Efficient application to the exposed skin before work of a protective substance suitable to the irritant concerned such as silicones, or paraffins with cetostearyl alcohol against water-soluble irritants kaolin with hard soap glycerin bentonite and stearic acid against oils and solvents and stearic acid with glycerin, casein and sodium alginate against dust.<sup>1</sup> Use of only the mildest cleansers after work, such as vegetable oils, liquid paraffin, good soaps. Special soaps in certain industries, e.g., containing 5% sodium sulphite to neutralize formaldehyde. T.N.T. and tetryl, or 10% sodium thiosulphate to neutralize mercury fulminate. Teaching both managers and workers that dermatitis is not an infection and that hard scrubbing strong soaps and antiseptics are the worst possible treatment. Education of the workers in prophylaxis including a warning of the increased risks of working when ill or insufficiently recovered from illness.

<sup>1</sup> For details of some typical barrier creams, see *Lancet*, 1934, 2, 976.

An important form of sensitisation dermatitis is that due to the dye paraphenylenediamine. This is used to dye furs and also to dye the hair. If the dye is not properly removed from the fur it is liable to cause a dermatitis on the wearer's neck after she has worn it for a longer or shorter period. The dermatitis is characteristically erythematous-vesicular situated on the sides of the neck and chin, and is always made worse by wearing the fur. Almost invariably the causal fur is that known as "beaver-coney" i.e. rabbit clipped and dyed brown to resemble beaver (vide Roxburgh, *Brit Jour Derm & Syph.*, 1935 37 pp 126-132).

The dermatitis caused by paraphenylenediamine when used as a hair dye usually starts on the sides of the face or neck, but it may spread all over the body. Some persons are very susceptible to this dye, others are resistant but may on occasion lose their resistance so that a test should be made on each occasion that the hair is to be dyed by painting on some of the dye behind the ear and covering it with strapping for twenty four hours, to make sure that no reaction is likely to follow the dyeing of the hair. Lipstick sometimes causes swelling and exfoliation of the lips due, usually to the red dye especially di- tri- or tetra bromofluoresceine (Zakon *et al Arch. Derm. and Syph.*, Chicago 1917 58 499). A number of cases have been described where dermatitis was due to nail varnish. The dermatitis occurred not alongside the nails but on the eyelids or other parts of the skin. The cellulose nitrate the solvent or the plasticiser were usually the culprits. Many cases of dermatitis have arisen from contact with india rubber in the form of anti gas respirators, rubber gloves, rubber corsets and occasionally even condoms. The irritant is usually either an accelerator antioxidant, or a plasticiser incorporated in the rubber to prolong its useful life. Dermatitis has also been occasioned by chemical substances introduced into furs and fabrics to improve their texture, feel appearance or properties. (Cox H. E. 1911 *Proc Roy Soc. Med.* 35 27) In the army in the 1939-45 war many cases of dermatitis were due to woollen khaki cloth in some cases the dye and in others the

wool itself seemed to be the irritant. Artificial tortoise-shell spectacle frames occasionally cause dermatitis where they touch the skin. By far the commonest type of sensitization dermatitis seen to-day however is that due to nickel in suspenders, earrings, brassiere clips, etc. It usually begins under one or more of the



FIG. 30 Suspender dermatitis (nickel sensitivity).

suspenders, spreads to involve other sites in contact with the metal and sooner or later also causes an eruption in certain areas not in contact, notably the antecubital fossae and upper eyelids.

Certain drugs applied externally frequently cause a dermatitis. Common examples are the sulphonamides penicillin strepto-

mycan, chlorpromazine, antihistamines and the benzocaine group of local anaesthetics. Mercury iodine and sulphur are less common causes, but it is probably true to say that practically every known medicament has produced a dermatitis in someone at some time. Depilatories (barium or strontium sulphide) often



FIG. 31.

Dermatitis due to india-rubber finger-stalls. Note that the little fingers are unaffected; stalls were not worn on these.

cause dermatitis of the face or axillae and "anti-sweating" or deodorant lotions (aluminium chloride or formaldehyde solutions) sometimes cause it in the axillae.

The resin contained in adhesive strapping very commonly causes irritation of the skin in sensitive persons. This can sometimes be prevented by varnishing the skin with Tinct. Benzoin Co. before applying the strapping.

TREATMENT of dermatitis should be on the same lines as that of eczema (p. 330) but in cases due to an external irritant it is of course essential to stop further contact with this and to avoid such contact in the future. Some patients by graduated

exposures become able to resist an irritant and can resume the work which caused the dermatitis, but the majority have to change their occupation. In cases of lip-stick dermatitis after cure by the giving up of lipstick and the application of hydrocortisone ointment for a few days a non-allergic lipstick should be used (Leschner, Queen). Similarly in cases of suspender dermatitis, plastic clothing accessories should be worn and nickel plated jewellery avoided.

### DERMATITIS ARTEFACTA

Eruptions are often produced artificially by patients. The objects may be to excite sympathy to get into hospital, to draw compensation, or to avoid work or military service. Cases also occur in which it is difficult to see what advantage the patient expects to gain. Patients with dermatitis artefacta are usually hysterical young women who had some perfectly genuine skin eruption to begin with, and who then, liking the sympathy and attention they got, or perhaps because they did not get as much as they considered their due, began to aggravate the existing eruption or to produce new lesions, by scratching with the nails, rubbing with the forefinger or by the application of irritants such as carbolic acid lysol strong alkalis, blistering fluid, etc. Such patients usually have anaesthesia of the bulbar conjunctiva and of the soft palate and suggestion anaesthesia to pin pricks of "stocking" and "glove" distribution. Many such patients seem really to be unaware that they produce the lesions themselves, and some of them almost appear to have a dual personality (MacCormac *Brit Jour Derm. & Syph.*, 1926 38 p 31). In other less common cases the lesions are produced quite deliberately and consciously for the purpose of malingering. Usually self inflicted lesions are easy to recognise, but sometimes their diagnosis is most difficult.

The points to be looked for are

- 1) The lesions do not correspond to those of any known skin disease

They have a bizarre appearance, being parallel lines,



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rectangular figures, or sharp-edged lesions of some other type on otherwise normal skin.

3 It is sometimes possible to see where a caustic liquid has run downwards from the lower edge of a lesion.



FIG. 22.

Dermatitis artefacta. Neurotic Excoriations.

4. The lesions are always in positions which can be reached by the patient's hand, usually the right hand. They are often, therefore, situated on the left arm or forearm.



FIG. 22.

*Dermatitis artefacta* produced by painting with nitric acid.

It is usually very difficult to persuade the relations and sometimes even the patient's own doctor that the lesions are self-produced.

**TREATMENT** Such patients are best removed from too sympathetic relatives and friends and placed in a hospital ward or nursing home where they can be watched. Even so it may be extremely difficult to catch them in the act of producing the lesions, and they are remarkably skilful at concealing small bottles of corrosive liquids. The affected part should be

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covered with an occlusive dressing which cannot be removed by the patient herself without detection, such as Unna's zinc



FIG. 34.  
Dermatitis  
arterialis  
Left Breast.

gelatine dressing or a plaster of Paris case. After a week or so it will be found that the lesions under the dressing have healed although possibly new ones may have appeared at the top and bottom edges. Psychiatry may be required.

### NAPKIN ERUPTIONS

Napkin eruptions are common in babies on the area covered by the napkin viz buttocks, small of back inner thighs, groins, perineum and genitals. They may be due in well-cared for babies to soap and soda being left in the napkins after washing. Babies napkins should not be washed with soda, and in any case should be put through two changes of clean water before drying. In babies who are allowed to remain in wet napkins the dermatitis may be due to the decomposition of the urea in the urine into ammonia by a urea splitting organism present in the faeces. In others again the eruption may be due merely

to maceration and infection of the skin as a result of prolonged contact with urine and faeces, especially if the napkin is enclosed in a pair of the popular waterproof drawers which convert it into a sort of faecal poultice. The trouble is particularly liable to occur in infants with diarrhoea where the stools contain digestive ferments.



*Dr. Adamson, Chm.*

FIG. 23.

Napkin Eruption. Note severe affection of prominent parts and sparing of flexures.

**CLINICAL FEATURES** The eruption is a bright erythema limited to the napkin area and most pronounced on the prominences in this area. The flexures, which escape actual contact with the napkin are spared. There may be sore places on the heels where they are in contact with the outside of the napkin. The eruption on the prominent parts may proceed to shallow ulceration. The baby is otherwise healthy and presents no eruption elsewhere. As Lindsay Batten has pointed

out, one can often diagnose the cause of a napkin eruption from its distribution those due to soda being all over the napkin area those due to urine being mainly on the inner sides of the thighs, and those due to faeces mainly about the annus.

The eruptions which are liable to be confused with these napkin erythemas are congenital syphilis, thrush, seborrhoeic dermatitis and impetigo. In *congenital syphilis* (p 269) the eruption is papular of a browner colour and extends into the



FIG. 26.

Napkin Eruption. "Vacciniform" type.

flexures. The baby usually has a papular rash also on the central parts of the face and a maculo-papular one on the palms and soles. Other signs of congenital syphilis are probably present also, such as a wirenéd appearance snuffles hoarse cry with possibly enlargement of the liver and spleen and tenderness of the ends of the long bones due to epiphysitis. *Thrush* (p 153) due to infection of the skin with *Monilia* (*Candida*) *albicans*, usually appears in babies which have had a thrush infection of the buccal mucosa. It appears as oval reddish macules tending to peel at the edges, the free edge of the peeling horny layer being

directed towards the centre of the patch. *Seborrhoea dermatitis* (p. 393) forms red sheets of eruption involving the flexures of the napkin area and possibly the axillae also. Beyond the edge of the red sheet are outlying red follicular papules.

*Impetigo* (p. 182) may be superimposed on either a napkin eruption or a seborrhoeic dermatitis. The lesions are flaccid clusters which readily rupture, leaving a raw weeping surface with a loose edge of horny layer at the periphery. The area involved may be large and there may be typical impetigo lesions elsewhere or on the mother or nurse.

**PROPHYLAXIS.** Napkin dermatitis can be prevented by avoidance of alkali in the napkins, by boiling them frequently and by always changing them as soon as they are soiled. The skin should be washed with a super-fatted soap and protected with a baby cream or baby powder. If the napkins smell ammoniacal, they should be well washed, thoroughly rinsed to remove every trace of soap and boiled daily. Before being dried, they should be soaked for three minutes in a solution of "Hoccol ordinary" (Bayer) 1 drachm (4 c.c.) in 2 quarts (2.27 litres) of water. This is sufficient for only six napkins as the chemical is adsorbed on to the napkins and so removed from the solution. After soaking, the napkins should be wrung out and dried. This treatment will retard the growth of the urea-splitting organisms.

**TREATMENT.** If a dermatitis develops, the boiling and soaking of the napkins should be continued and the skin protected by a silicone barrier cream (Siopel, Vasogen) or zinc and castor oil cream.

If the dermatitis should be severe, the napkins should be laid under and not fastened on to the child. In the presence of a dermatitis, washing with soap will probably irritate the skin and, in that case, the skin should be cleaned with liquid paraffin. Should the dermatitis become infected a local antibiotic should be used, e.g. 0.5% neomycin lotion.

## CHAPTER VII

### DRUG ERUPTIONS

A NUMBER of eruptions are produced in susceptible persons by the ingestion of drugs. In some cases there appears to be an idiosyncrasy and small doses produce the eruption, in others overdoses or administration over a prolonged period are necessary before the eruption appears. Sometimes the eruption is the only symptom, in other cases there are general symptoms also such as fever furred tongue congested fauces, nausea, vomiting diarrhoea, headache, giddiness etc. Renal disease as interfering with the elimination of drugs appears to be a predisposing cause of drug eruptions.

The site of drug eruptions is most commonly on the trunk, neck, shoulders upper arms and thighs. In type they are usually erythematous, but they may be urticarial, vesicular bullous, pustular etc. The following list (after MacLeod) gives the principal types of eruption which occur and the drugs which may cause them.

TABLE III  
(After MacLeod)

Erythematous.—Papular	Antipyrin aspirin belladonna bismuth gold iodides mandelic acid phenacetin phenobarbitone sulphonamides, etc
Macular and Patchy	Animal serum amidopyrin antipyrin arsenic aspirin belladonna bismuth bromides chloral codeine gold io- dides morphine opium phenacetin phenobarbitone potassium chlorate qui- nine salicylates sedormid sulphona- mides turpentine
Morbilliform	Animal serum antipyrin arsenic bella- donna meprobamate me-antoin pheno- barbitone phenylbutazone sulphona- mides sulphonal triethione turpentine
Scarlatiniform	Animal serum antipyrin arsenic bella- donna chloral codeine glutethimide ( Doriden ) hyoscine mandelic acid,

TABLE III—Continued

opium phenobarbitone pilocarpine quinine streptomycin sulphonal turpentine.

**Exfoliative Dermatitis** (Secondary Erythrodermia) Chrysarobin and derivatives and tar externally Arsenic and gold by injection. Streptomycin. Sulphonamides.

**Urticarial.** Animal serum arsenic aspirin bromides iodides mercury opium penicillin phenacetin phenobarbitone phenolphthalein quinine salicylate of sodium santonin sulphonamides thiocarbollin dione turpentine etc

**Vesicular** Antimony antipyrin arsenic bromides chloral gold iodides phenobarbitone phenolphthalein quinine sulphonamides; turpentine etc

**Herpetic (Herpes zoster or Herpes simplex)** Arsenic Bullous. Antipyrin bromides chloral iodides opium phenobarbitone quinine salicylates etc.

**Polymorphous eruptions of the erythema multiforme type** Animal serum analopyrin antipyrin chloral iodides opium potassium chlorate sulphonamides etc

**Festular** Antimony antipyrin arsenic bromides iodides opium salicylates turpentine. (Of these the most common causes are bromides and iodides.)

**Acneiform.** Antipyrin arsenic bromides chloral iodides opium

**Ulcerative** Arsenic bromides chloralhydrate iodides.

**Condriomatous or Anthracoid.** Bromides iodides.

**Gangrenous.** Arsenic iodides quinine

**Purpura and Petechial** Animal serum antipyrin aspirin bromides carbosmal ergot iodides meprobamate potassium chlorate quinine salicylates sedormid sulphonal sulphonamides

**Keratotic** Arsenic bismuth gold

**Pigmented** Arsenic carbosmal gold silver nitrate phenolphthalein.

The commonest drug eruptions are those due to penicillin





FIG. 37

Arsenical hyperkeratosis. Patient took 20 to 30 minims of Fowler's solution a day on and off, for two years but had taken none for two years before photograph was taken.

streptomycin and carbromal. Those due to arsenic, gold, bromides and iodides, mepacrine sera and sulphonamides, though less frequently seen nowadays, are important because of their severity

**Arsenic.** Prolonged ingestion of inorganic arsenic, such as used to be given for psoriasis and dermatitis herpetiformis, and added to bromides for epilepsy and iron for anaemia, can cause a cutaneous change many years after the patient has ceased taking the drug. Such cases are, therefore still seen and show multiple small warty excrescences known as punctate keratoses, notably on the palms and soles, but also on the trunk and limbs. Pigmentation also occurs and this is most marked in situations which are normally pigmented or which are exposed to friction but it also occurs on the trunk in a mottled fashion known as raindrop pigmentation. Finally multiple basal cell and squamous cell epitheliomata may develop in the skin. In a proportion of these cases, squamous cell carcinomata also occur in internal organs, notably the bronchus.

**Organic arsenicals** can cause exfoliative dermatitis. This usually occurs towards the end of a course begins in the flexures and spreads quickly to involve the whole of the skin, causing erythema, thickening and scaling. It is a severe and potentially fatal condition (see Chap. XXIII p. 452). These drugs also cause an evanescent erythema on the 9th day of treatment which disappears in a few days without therapy and does not recur with further doses of the drug (Millian's 9th day erythema). There is, however another type of early punctate erythema which appears on the second or third day after an injection and is accompanied by much irritation. This type may be the precursor of true arsenical dermatitis if arsenic is persisted in, but it may be impossible to differentiate on examination from ninth day erythema. True arsenical dermatitis develops into a more or less severe exfoliative dermatitis which may prove fatal from broncho-pneumonia, toxæmia, intestinal hæmorrhage or sepsis. The incidence of severe dermatitis following arsenical injections varies but may be taken as something of the order of one case in every 2000 treated. Predisposing causes are focal sepsis and pre-existing dermatitis or eczema.

**PROPHYLAXIS** In many of the cases of arsenical dermatitis which I have had to treat close questioning of the patient has revealed the fact that he or she had one or even two or three,

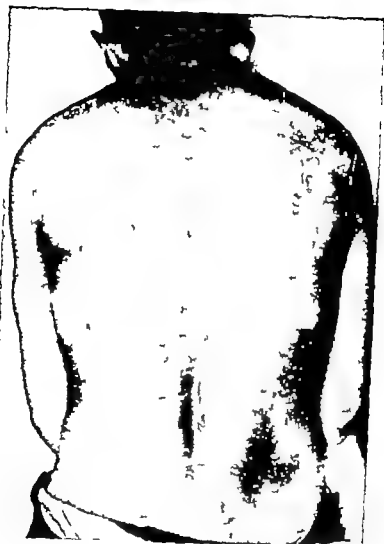


FIG. 34.

*Dr. Adamson's Case.*

Arsenic I Pigmentation. Note the white raindrop spots.

further injections after the rash had first appeared on the arms. Many cases of arsenical dermatitis would be avoided if all patients having arsenobenzene injections were questioned *before*

every injection as to whether any rash had appeared on the forearm after the previous injection. If any rash did so appear the arsenic should be withheld for some weeks and then tried again in very small doses, using a different brand of "914". Keeping the patient saturated with vitamin C has been said to reduce the likelihood of arsenical dermatitis.

**TREATMENT** The patient suffering from exfoliative dermatitis should be treated in bed. Only calamine lotion or equal parts of olive oil and lime water are the most soothing local applications, although any bland emollient cream may be used. Steroid hormones are given by mouth prednisolone mg. 20-40 daily usually producing complete cure in 4-6 weeks. For this reason dimer caprol (B.A.L.) has been largely superseded since, to be effective, it has to be given within a few hours of the last dose of the arsenical. It is given by deep intramuscular injection in doses of 8 mg. per kilogram of body weight (200 mg. or 4 c.c. for an average adult) four hourly for the first two days and then at gradually increasing intervals for ten days. The drug may cause nausea and vomiting, lachrymation, salivation and a burning feeling, and local abscess formation may occur.

Gold given by injection whether for rheumatoid arthritis, lupus erythematosus or tuberculosis is the cause of skin or mucous membrane lesions in a considerable proportion of patients, from 10-30% in different series. It may also cause damage to the liver, kidneys, alimentary canal, central nervous system and haemopoietic system. The skin eruptions may be erythematous, scaly resembling pityriasis rosea, pruritic, papular, Eichenoid, pustular, pigmented, purpuric, urticarial, vesicular or bullous, and a proportion proceed to exfoliative dermatitis which may be fatal.

Prophylaxis depends on careful examination of patients and enquiry before each injection as to signs or subjective symptoms following the previous injection, examination of urine for albumin, blood and casts, examination of blood for leukopenia and eosinophilia (over 7%) and keeping the dosage of gold low as regards both individual injections and total in a series. Not more than 40 weekly injections should be given at any one time,

and each dose should not exceed 0.025 g. Sodium aurothiomalate (myocrisan) is the most suitable preparation.

The treatment of gold dermatitis is on the same lines as that of arsenical dermatitis including the use of steroid hormones or dimercaprol.



FIG. 30.

Gold Dermatitis. Author's case from *Bril. Jour. Derm. & Syph.* 1936, 48 137

**Bromides.** Bromide eruptions may appear after a single small dose in a susceptible person, but more commonly do so after the drug has been taken for some time. They may occur

in a breast fed infant if the mother is taking bromide. Usually they occur on the face, shoulders and legs. They are pustular and resemble a pustular acne but there are no comedones. A fungating papillomatous type of bromide eruption sometimes occurs with beads of pus exuding from between the papillae. Such lesions are often covered with a brownish or greenish crust and are most common on the legs. Bromide eruptions



FIG. 40.

Dr. Johnson Case.

Bromide Eruption. Fungating Papillomatous Type.

often take a long time to disappear after the drug has been stopped so that little help in diagnosis can be expected from stopping the drug and watching results. Bromine may however be detected chemically in the urine. Bromides also pro-

duce less characteristic eruptions, such as erythema, urticaria, vesicles, bullae and varicelliform lesions.

**Iodides** Iodides commonly produce pustular acneiform



FIG. 41.

Iodide Eruption. Result of taking a proprietary blood mixture for Rosacea.

lesions without comedones, usually on the face, neck and shoulders. Vesicular and bullous eruptions may also occur especially in patients with dermatitis herpetiformis renal disease, or infective endocarditis. Other types of iodide eruption resemble the fungating papillomatous lesions produced by bromides, tuberculous masses resembling gummata, sarcomata or the tumours of mycosis fungoides. Such lesions, when they occur usually do so on the face forearms, hands, legs, or feet, but they may appear anywhere. In cases where syphilis is suspected the

lesions are often mistaken for gummata and the iodide is continued or increased with unfortunate results. Assistance in the diagnosis of iodide eruptions is sometimes obtained from the simultaneous presence of symptoms of iodism, *e.g.* corvina, salivation, gastro-intestinal irritation, etc. Iodine may also be detected in the urine by chemical tests.



FIG. 4.  
Phenolphthalein eruption.

**TREATMENT OF BROMIDE AND IODIDE ERUPTIONS.** The first step is to stop the drug and the second to try and hasten the elimination of it by giving sodium chloride either by the mouth  $\text{xx t.d.s.}$ , in salol or gelatine capsules (Stevenson) or intravenously 100-400 c.c. of a decinormal or a physiological



grey pigmentation occurred affecting the nail beds, hard palate gums and cartilages of nose and ears. The mucosal lesions resembled those of lichen planus but typical polygonal lichen planus papules on the skin were absent and the glans penis was not affected. This might be the first eruption but usually followed the patchy eczematoid type (3) Generalised exfoliative dermatitis. This usually followed type 1 or 2 but might be primary in which case it might be acute, oedematous and pyrexial.

Pigmentation was sometimes very persistent and in some cases there was a fine reticular atrophy. Hair and nails were often shed but always grew again. Though the three types mentioned were the commonest the disease might resemble almost any known dermatosis.

Treatment was unsatisfactory but nowadays the condition can be controlled by steroid hormone therapy.

Penicillin preparations applied externally set up a contact dermatitis at once in a few patients but much more commonly do so after a week or ten days application. In some cases this is due to sensitisation to the penicillin itself but often to the vehicle in which it is applied. If it is considered necessary to continue the penicillin in such cases patch tests (p. 330) should be done with (a) penicillin solution and (b) the vehicle without penicillin in order to decide upon the culprit. Because of the risk of sensitising a patient to penicillin and so prejudicing his chances in some future serious infection it is now generally considered unwise to use penicillin preparations externally.

After injections of penicillin especially after very large doses, some patients develop urticaria, joint pains, swelling of lymph nodes, fever and generalised dermatoses of various types. The urticaria may come on immediately or not for several weeks, and it is sometimes very severe and persistent. It should be treated by stopping the penicillin if this is safe and by the use of anti histamine drugs adrenalin etc (p. 301). Cortisone (p. 36) will relieve the symptoms in severe cases. Chronic eczema may be aggravated by penicillin injections. Some degree of reaction to penicillin injections probably occurs in 5 to 10% of patients receiving them. More than fifteen cases of so-called

death following injections of penicillin were reported in the years 1950-54. These were apparently due to anaphylactic shock. (*Lancet* 1954 1 31)

/ Phenolphthalein is the commonest cause of the so-called fixed drug eruption. In this condition one or more well-defined circular patches of dusky erythema appear usually on the trunk or proximal portion of the limbs, a few hours after taking the drug and last for 2-3 days. On subsequent occasions the lesions recur in exactly the same sites, although fresh lesions may occur and old lesions spread slowly peripherally. The affected areas are usually marked by increasing pigmentation between attacks. Mucosal lesions have also occurred in the eye, mouth and urethra.

**Serum eruptions** Between the seventh and twelfth days after an injection of an animal serum, e.g. anti-diphtheritic, anti-tetanic etc. many patients develop a widespread erythematous and/or urticarial eruption. This is often associated with a rise of temperature to 101-102° headache, joint pains and often transitory albuminuria. The rash often persists for two to six days. It may be relieved by one or other of the antihistamine drugs (v. page 301) or by subcutaneous injections of adrenalin  $\text{M} \times$  (0.5 c.c.) but frequently requires steroid hormone therapy. As local applications to relieve the itching, calamine lotion with 2% carboic acid or some other antipruritic (p. 24) should be used.

Streptomycin, nowadays used chiefly for the treatment of tuberculosis, causes drug eruptions in about 5% of cases. The commonest types are erythematous, maculopapular and urticarial and occur between the fourth and fourteenth day of treatment. Exfoliative dermatitis can also occur coming on later rarely before the end of the third week, and may be severe. Systemic symptoms are common, usually fever and arthritis, but hepatitis, nephritis and encephalitis have been described. Treatment consists of antihistamines for the mild cases and steroid hormones for the severe ones. Active desensitization has proved successful, both for sensitization following injection of the drug and when topically applied, the latter most frequently being seen in nurses giving the injections. The incidence of

this, however has been very greatly reduced by the wearing of rubber gloves and by forbidding the expressing of air bubbles from the syringe prior to injection, a practice which caused a fine spray of streptomycin to be blown up into the nurse's face. Desensitization is carried out by daily subcutaneous injections of gradually increasing amounts, beginning with 1 mg



FIG. 44

Sulphonamide dermatitis, due to sulphanilamide internally

**Sulphonamides** Eruptions due to *internal administration* usually appear about the end of seven days treatment but they may do so within a few hours of the first dose or not for a week or more after the end of the course. They are usually

accompanied by fever and malaise, but may be afebrile. They may be localised or generalised and may be associated with adenitis, stomatitis and conjunctivitis. There may be some eosinophilia. In type they may be erythematous, macular morbilloform, urticarial, papular vascular purpuric or exfoliative. In 2-4 % of patients the skin becomes light-sensitive. Sulphathiazole may produce lesions like erythema nodosum (p. 263)

The drug should be stopped and large quantities of water given. Lead and calamine lotion or a dusting powder may be applied. Antihistamines are helpful and steroid hormones may be necessary in severe cases. If a patient has reacted to one sulphonamide, they frequently are found to be sensitive to all members of the group.

Because of the frequency of subsequent dermatitis, the local application of these drugs has been abandoned.

**Cross-sensitization** Some patients develop a sensitivity to more than one drug. In certain cases, this can be explained by the fact that they react to a particular chemical group occurring in a number of chemically related compounds. The best example of this is the para-aminophenol group found in certain sulphonamides, the benzocaine group of local anaesthetics, para-aminosalicylic and para-aminobenzoic acids, para-phenylenediamine (the active ingredient of certain hair dyes) some of the azo-dyes, etc. Patients are sometimes seen who have reacted to a number of these substances throughout their lives. Care should be taken, therefore, in giving other drugs of this group to an individual known to be sensitive to one of them.

males and immature females. After impregnation the females, now mature again start the cycle which, from egg to mature female takes 10-14 days. There is such a heavy mortality however that well under 10% of the eggs ever give rise to adult mites and it is often several weeks before adults of the second generation make their appearance.

**INFECTION** Fairly close and prolonged contact is usually required for the transmission of scabies the *scarus* being a slow mover. Its best speed is about 1 inch per minute on a warm skin and it takes about 1 hour to bury itself. Below 16° C. (61° F.) it is completely paralysed by cold and even up to 90° C. (68° F.) practically no movement takes place. The disease is usually contracted in bed from an infected bedfellow though children seem often to get it by holding hands or playing together. The usual social contacts between adults are unlikely to spread scabies. K. Mellanby's experiments make it seem unlikely that bedclothes often convey the disease though they undoubtedly can do so. Adult females are the usual agents in transmission although nymphs may transfer the disease occasionally.

**CLINICAL FEATURES.** Mellanby has shown that on a first infection with scabies, a patient does not begin to itch for a month or more during which time he is unconscious of his disease but just as infectious, at least in the latter part of that time, as a patient who is itching violently. After about a month the patient seems to become sensitised to the *scarus*, erythematous patches appear round the burrows follicular papules appear and the patient begins to itch. This itching is intermittent and usually worst at night coming on as soon as the patient has got warm in bed. The resulting scratching leads to abrasion of the skin and often to infection with staphylococci or streptococci leading to widespread impetigo ecthyma and boils. Sensitisation to the *scarus* may give rise to a perfectly genuine urticaria or eczema and the irritation may be ascribed to this the presence of scabies being missed. Any of these complications may continue long after the scabies has been cured and even in their absence the irritation may persist for

some weeks after the last scarus is dead. Conversely a few patients with obvious scabies appear not to itch at all.

Mellanby has found that when a patient has once had scabies and so become sensitised a subsequent reinfection causes irritation within a few hours. This often leads to cure of the new infection at once by scratching out the parasite.

The physical signs of scabies are the burrows, which are pathognomonic and a red follicular papular rash which is produced partly by the presence of the immature scari in the hair follicles, partly as a result of sensitisation and partly by scratching. This rash is most pronounced on the abdomen, around the axillae and on the inner sides of the thighs.

The sites on which burrows are most commonly found are the anterior aspect of the wrist, the ulnar edge of the hand, between the fingers, the point of the elbow the anterior axillary fold, the lower part of the buttocks, the inner sides of the feet the penis in men, and around the nipples in women. At the inner end of the burrow a small vesicle may sometimes be seen this is situated in the prickle-cell layer beneath the burrow and the scarus should be looked for *beyond* the vesicle. On the penis the burrows are usually inflamed and appear as oval or elongated red infiltrated lesions.

The numbers of adult females present in different cases of scabies vary greatly. In nearly 900 patients Mellanby found that more than half of them harboured only from 1 to 5 adult females each, while only 3% of them had over 50. The greatest number found in any one case was 511 and the mean was only 11.3. Presumably patients harbouring large numbers of mites are proportionately infectious.

Clinically severe i.e. secondarily infected cases usually have very few scari and these generally only on areas free from secondary infection.

The mite population increases up to 100 days from infection when it reaches a maximum and then there is a catastrophic fall in numbers and sometimes spontaneous cure. Symptoms may be worse after the fall in population.

The rare condition Norwegian or crusted scabies is characterised

by extensive crusting and scaling and by the enormous numbers of acari present even in the finger nails. It is probably the result of neglect and is comparable to *sarcoptes mange* in animals where many thousands of parasites occur on a single host.



*Dr W. Herbert Brown's Case*

FIG. 47

Scabies. The lesions on the palm are typical burrows but they are seldom seen in such numbers.

**DIAGNOSIS.** The first point to remember is that scabies is no respecter of persons. When confronted with any itching eruption in a patient in any social position one should invariably make certain that the disease is not scabies before even considering any other diagnosis. Scabies is constantly missed in patients of good social position because it is not thought of

The recognition of burrows makes the diagnosis certain enough for practical purposes, but it can only be considered absolute if an acarus is extracted and identified under the microscope. This is quite easy provided the acarus can be seen at the end of the burrow with a hand lens. If she cannot be seen there is no use trying to pick her out. When the minute brown and white speck which is the acarus is seen at the inner end of the burrow a sharp needle should be held parallel with the skin surface and used gently to lift off the horny layer above the acarus. She will then in most cases adhere to the point of the needle and can be transferred to a microscope slide and mounted in liquor potassae. If no burrows can be positively identified, but if an itching eruption is most pronounced on the areas above mentioned as characteristic sites for scabies burrows, or even on two or three of them, then it is usually wise to treat the patient for scabies, as no harm will be done even if the diagnosis is incorrect. Children with widespread impetigo are usually found to have either scabies or *lichen urticatus* (p. 303).

**TREATMENT** The traditional treatment of scabies is a hot bath using soft soap and a nail brush to lay open all the burrows, followed after drying, by a thoroughunction with two ounces of Ung. Sulphuris B.P. (10%) over the whole body below the neck. The unction is repeated without the bath on the two following nights and a final bath is taken on the fourth morning. After this clean clothes are put on and all the under-clothing, outer-clothing and bedclothes are disinfected by heat or otherwise. This method if properly applied will cure 100% of cases of scabies. Its drawbacks are that the sulphur ointment is messy, has a characteristic smell and rather easily gives rise to dermatitis in susceptible people, although if the applications are limited to three the percentage of patients developing dermatitis will not be large and if the ointment is made with a vanishing cream base it is much pleasanter to use and just as effective. For children the ointment should be diluted to 5%.

The usual treatment now is by an emulsion of benzyl benzoate such as that of the National Formulary made with emulsifying



wax or the following given by Mellanby (*Scabies* Oxford Univ Press 1943 p 76).

(a) Benzyl benzoate	200 c.c.
Stearic acid	20 gm.
Triethanolamine	6 c.c.
Water to produce	1000 c.c.

Heat together the benzyl benzoate and stearic acid until the latter is dissolved. Mix the triethanolamine with the water and then pour this into the warm benzyl benzoate-stearic acid mixture and stir.

This preparation has the advantage that it appears impossible not to make a good emulsion and the liquid is much the least painful of all the emulsions on a tender and abraded skin.

The patient should be dried after his bath and the emulsion thoroughly applied all over the body below the neck using a flat paint brush two inches wide. He is then left in a warm room until he is dry which takes 10 to 15 minutes. About 2 ounces (60 c.c.) of the emulsion are required for each treatment. Benzyl benzoate emulsions need not be diluted for children.

Mellanby and his collaborators have found that whether a patient is scrubbed or not before treatment with either Ung Sulphurum or benzyl benzoate lotion makes practically no difference to the percentage of mites killed by one application and the official recommendation therefore is that nothing harder than a rough flannel need be used in the preliminary bath. While it may be true that it is not necessary to lay open all the burrows by scrubbing in order to effect a cure, it is exceedingly difficult to tell whether a patient is cured or not if there are a number of unopened burrows containing acari still viable. In his experiments Mellanby judged of cure by extracting all visible acari and examining them on a warm slide to see whether or not they moved. If not and if their suckers were distended he judged them to be dead. This method however is obviously impossible in ordinary practice and I prefer therefore to have the patient thoroughly scrubbed as a preliminary to treatment.

While one thorough application of benzyl benzoate emulsion

will cure over 95% of cases Mellanby recommends a second application within 8 days. In order to save time and get the patient cured quickly I am in the habit of prescribing three treatments with benzyl benzoate on three successive days the patient wearing his infected clothing until the series is completed when he takes a second bath and puts on clean clothes.

Another preparation which may be relied on to cure scabies if properly used is Ung Potassi Polysulphidi B.P.C. This, while it will often cure scabies with one application, is liable to produce dermatitis if used more than once.

A very useful preparation especially in children and in those with much eczema, dermatitis or secondary infection is Balsam of Peru 3 i. Zinc cream (p 28) 3 i. (12%) This may be applied if necessary for a week.

Mesulphen (Blutagal) is a yellow oily liquid which is extremely effective even in a 10% solution in liquid paraffin and is non irritating. It is, however expensive.

Patients must be warned that if itching persists as it frequently does, after treatment has been completed they must not continue the treatment or they will only cause dermatitis. They should be advised not to bath for a few days and to apply twice daily either an oily calamine liniment or a zinc cream containing 12% of liq. pure carbonis (p 28).

A patient who has been applying sulphur ointment casually to various parts of the body for a long time will probably have both scabies and sulphur dermatitis when first seen. Such patients should be treated by either benzyl benzoate emulsion or balsam of Peru 1% in zinc cream.

In patients with extensive secondary infection either this or the scabies may be treated first according to circumstances (p 189).

With regard to the disinfection of clothing. It appears that the benzyl benzoate or sulphur ointment on the skin is sufficient to kill all mites on the underclothes and that ironing with a hot iron is all that is required for the outer clothing or blankets, while ordinary laundering will sterilize the sheets. So

that unless there are special reasons routine disinfection is not now regarded as necessary by the Ministry of Health.

The acarid dies in less than ten minutes at 50 C. (120 F) regardless of the humidity so no great heat is required to disinfect clothing. The acarid lives longest at 13° C (55 F) when the air is moist, but even under these conditions it dies in less than two weeks. It dies in less than two days under the conditions found in a drying cupboard.

What is infinitely more important than the disinfection of fomites is the treatment of all personal contacts of the patient whether they itch or not, because as shown above, patients may have scabies and be infective for 4 to 6 weeks before they are conscious of any itching.

Scabies may be made notifiable under the Scabies Order 1941 and the M.O.H. is empowered to require other inmates of a house where a scabies patient has been living to submit themselves to examination and if necessary to treatment. He is also empowered to require disinfection of clothing, etc. if considered necessary.

**PROPHYLAXIS** Mellanby found that the use of "Tetmosol" soap in an asylum where scabies was endemic prevented new cases arising among those using the soap and cured many patients who were originally infected. Tetmosol soap contains 10% of tetra ethylthiuram monosulphide.

**Animal scabies** (*Sarcoptic mange*) not infrequently attacks human beings especially the owners of affected lap dogs or cats, and grooms attending mangy horses. The burrows characteristic of human scabies are not found, the eruption being a small red papular rash which affects the arms or other parts which have been in contact with the infected animal. The best place to find the acarid is the point of the elbow. When the source of infection is detected and cut off the disease tends to die out of itself, and it is more easily cured with sulphur than is human scabies. Camel scabies has frequently caused trouble in troopers in camel corps and keepers in zoological gardens.

**Miscellaneous Mites.** Numerous other mites occasionally attack man and cause eruptions of an intensely irritating papulo-

vesicular or urticarial type. Among these are bird mites (*Dermanyssus*) which attack those who look after poultry and occasionally invade houses from nests deserted by starlings or other birds. Also the mites (*Pediculoides*) normally parasitic on moth larvae in grain or straw which often attack those who have to handle these things. The mites living on dried fruits and copra (*Carpoglyphus*) or on cheese (*Tyroglyphus*) cannot pierce the skin of man. The irritation which sometimes occurs

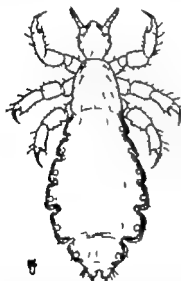


FIG. 46.

Head Louse. *Pediculus capitis*. Female. Dorsal View (20). Small figure indicates natural size. The Body Louse is similar but slightly larger.

(British Museum, Economic Series, No. 2. B. F. Cresson, 1912.)

in those handling such substances when heavily infested with mites appears to be due to an allergic reaction to the mites their corpses or their dried excrement. The primary infection in such cases is easily cured with sulphur or benzyl benzoate on removal of the source but the results of scratching may as in scabies, be more difficult to get rid of.

**Pediculosis** Three kinds of louse may infest the human body the Head Louse, *Pediculus capitis* the Body Louse, *Pediculus vestimentorum* or *Pediculus corporis* and the Crab Louse *Pediculus* or *Phthirus pubis*. They obtain food by puncturing the skin with a proboscis and sucking out blood. Human

beings may also be bitten on occasion by animal and bird lice. Lice are six legged, and are wingless insects. *P. capitis* and *P. corporis* are elongated in shape and about 3 mm long, *P. corporis* being the larger. *P. pubis* is shorter and as broad as it is long with large claws on its hinder legs with which it grasps the hairs.

These lice are all of a dirty greyish colour on Europeans.



FIG. 49

Two eggs-on-pub  
(“Kits”) of *Hem*  
Louse on hair  
(After Saberswood)

*Pediculus capitis* attaches its eggs to the hairs of the head. Each egg is contained in a minute, grey cup-shaped receptacle the nit, which is attached by a collar to the hair. It can be slid up and down the hair but cannot be pulled off unless slid up to the end. The eggs hatch in 6-16 days and the young lice become mature after three moults in a further 8-16 days (Buxton). Mellanby found that in ten industrial cities in this country about 50% of girls and 27% of boys between the ages of two and thirteen years had pediculosis capitis. From fourteen to eighteen years 37% of girls still had it. The incidence in country districts was only from 4 to 9% for both sexes. It is small wonder then that people otherwise clean occasionally contract this infection. Pediculosis is most common on the back and sides of the head. The irritation caused by the bites of the lice causes scratching, and this leads to infection of the scalp with pus cocci, resulting in impetigo and pustular lesions. These in turn lead to enlargement of the posterior auricular and cervical lymph glands. The

impetigo may extend on to the face and the pus infection may also be transferred to the eyes by the fingers. Impetigo on the scalp or back of the neck should always suggest a careful search for pediculosis.

**TREATMENT** Dr E. B. S. Scobbie found that one application of a 2% DDT emulsion kills all lice and remains long enough on the head (15 days) to kill all larvae as they hatch out, though

It does not kill the ova (Buxton) D.D.T. is not removed to any serious extent by washing. Benzyl Benzoate emulsion 25% as used for scabies, is also effective.

An excellent method (K. Mellanby) is to soak the scalp and hair thoroughly with methylated spirit 7 parts, water 3 parts. This may be poured repeatedly over the hair or the head may be covered with lint soaked in the mixture. The wet hair should then be covered for one hour with a rubber bathing cap. After removal of this the hair dries quickly and need not be washed. The nits should be removed by careful combing with a small-tooth comb. The impetigo if present must now be dealt with on the usual lines (p. 189).

Whitfield's method is to soak the hair thoroughly with hot 1-40 carbolic acid by pouring this repeatedly over the head and combing the hair to make sure it is thoroughly saturated.

The head is then tied up in a towel for one to two hours. After this the head may be washed or simply allowed to dry and the nits removed as above. These methods kill both lice and ova, and are therefore preferable to the traditional paraffin and *sassafras* which do not kill the latter. Finally it is important to sterilise the hat as well as the head.

A method for both prevention and treatment of pediculosis capitis which has been found very useful in the women's services is the application of organic thiocyanates and laurates e.g., "Lethane 381 special." For use this is diluted with 50% of liquid paraffin and scented with 2% of oil of citronella. From two to three drachms (8 to 12 c.c.) according to the size of the head and length of the hair is distributed with a teaspoon or fountain pen filler through partings to twelve points on the scalp six on each side. It is then spread by massaging with the fingers not by combing as it is wanted on the scalp not on the hair. A second application a week later gives a certain cure (Scoltée). The head should not be washed for 8 to 10 days after an application of lethane. Lethane is said to kill the ova as well as the adults.

*Pediculus corporis* (*P. vestimentorum*) lives on the under clothing to which it attaches its eggs, although these are

occasionally fastened to the lanugo hairs of the midline of the back, and in heavy infestations to the pubic and axillary hair also (H. W. Barber). The neck of the underclothing and the seams of this and of the trousers are the most favoured places. The life history is similar to that of *P. capitis*. The life of the adult is three to four weeks, during which time a female will lay from 200-300 eggs. *P. corporis* lives on the underclothing and turns to the skin only for food. Therefore it tends to bite those areas which are in close contact with the underclothes, e.g. the upper part of the shoulders and upper part of the buttocks.

The bite is visible as a minute red macule which irritates greatly. The linear scratch marks which patients with pediculosis corporis make on their shoulders with their nails are very characteristic and almost enough for a diagnosis. Pediculosis corporis is quite common in elderly people of good social position, and scratch marks on the shoulders in such patients should always lead to a careful search for parasites or ova on the underclothes. Pediculosis corporis, and the scratching to which it gives rise cause after a time thickening and pigmentation of the skin, the so-called Vagabond's Disease. No tolerance is developed to louse bites even after years of infestation (Buxton). Pediculosis corporis is of importance because typhus, trench and relapsing fevers are all transmitted by the body louse and pediculosis is always rife under conditions of overcrowding with limited washing facilities, such as occur in an army on active service, or in a population living in air-raid shelters. Typhus (due to *Rickettsia prowazekii*) and trench fever (*R. quintana*) are transmitted by the faeces of an infected louse getting into a scratch on the skin, relapsing fever (*S. recurrentis*) by the blood of a crushed louse infecting a scratch.

**TREATMENT** A hot bath followed by thorough powdering of the underclothing with a 10% D D T powder will rid the patient of lice. The impregnation of underclothing with 1% by weight of D D T will keep the wearer free from body lice though not from crab lice, for about one month in spite of weekly laundering. A powder consisting of 10% of D D T in an inert diluent proved in-

valuable in the army and in controlling louse infestation in civil populations during the late war e.g. in the Naples typhus epidemic in 1914. The powder is blown into the clothes with a blower at five sites without undressing the patient.

If D.D.T. is unobtainable a hot bath followed byunction all over the body of xylol 25% in equal parts lanoline and petroleum jelly or Ung. staphisagris or Ung. sulph. or Ung.  $\beta$ -naphthol 4% or 2% creosol solution and the shaving of any hair bearing nits will clear the patient. The disinfection of the underclothing is the most important part of the treatment. This is best carried out by heat or by steam but can also be effected by soaking in petrol, or in 5% cylin.

*Pediculus (Phthirus) pubis* lives usually on the pubic region but may be found about the anus and in the axillae, also on the chest or limbs of a hairy man and even on the eyebrows or eyelashes. Its characteristic attitude is with its claws firmly grasping adjacent hairs and its mouth parts buried in the skin. The nits are attached to the pubic hairs or the hairs of the other regions mentioned. They are darker in colour than those of *P. capitis*. The eggs hatch in 7-8 days and the duration of larval life is 13-17 days. *Pediculus pubis* is usually acquired in sexual intercourse but may be picked up from the seat of a water-closet. Its bite causes much irritation, and in cases of pruritus ani and vulvae *pediculosis pubis* should be excluded by careful examination before any other cause is thought of. *Maculae caeruleae* are bluish-grey macules  $\frac{1}{2}$  1 cm. in diameter found on the sides of the trunk and the inner surfaces of the arms and thighs in some fair-skinned individuals infested with *P. pubis*. They are supposed to be produced by a pigment injected with the saliva of the louse.

**TREATMENT** Rub the affected areas with a 10% D.D.T. powder or a 1% solution of D.D.T. in liquid paraffin. If these are not available benzyl benzoate emulsion 25% as used for scabies is effective. A simple and certain method is to shave off all the affected hair and then to apply 25% of xylol in equal parts of lanoline and petroleum jelly for a few days. The objection to this is the irritation caused by the pubic hair when it is growing again.



after shaving. Short of shaving the affected parts they may be thoroughly soaked with Ac. carbol. 1-40 or methylated spirit 7 parts water 3 parts, for an hour (rede P capitis, p. 144), and the nits then removed with a small tooth comb. The above ointment should then be applied for eight days in case any parasites or ova have been missed.

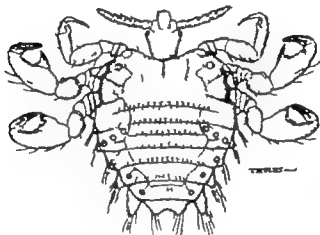


FIG. 50.

Crab Louse. *Phthirus pubis*. Dorsal View

**Dangers of D.D.T.** (*Dichlorodiphenyl Trichloroethane*). In the usual concentrations and amounts used as insecticides and parantides especially in powder form, D.D.T. is harmless to man and domestic animals, though some cats and dogs have died from its application, but absorption resulting from prolonged exposure to oily solutions or from intense exposure to concentrated solutions, e.g. in acetone, may cause toxic symptoms such as tiredness and irritability, heaviness and aching of the limbs, muscular weakness, fine tremors and finally respiratory failure. Chronic poisoning by small doses results in anorexia with damage to liver and kidneys.

In an African child of 1½ years who swallowed an ounce of a 5% solution of D.D.T. in kerosene the lethal dose was about 150 mg. per kilo. of body weight (Hill and Robinson).

Repeated application of strong solutions to the skin in animals causes acute inflammation oedema and sometimes haemorrhage. The lesions heal rapidly when the D.D.T. is removed (Crauford Benson).

**Fleas** Some fortunate individuals appear to be naturally distasteful to fleas while others possess or acquire a relative immunity to the effects of their bites. The site of a flea bite is



FIG 51.

Common Bed Bug. *Cimex lectularius*. (Female.) Dorsal View.  
Lens 1 scale indicates natural size.

(British Museum Economic Series, No. 1. B. F. Cresson 1917.)

shown by a minute haemorrhagic point in the centre of a red macule. The latter sometimes develops into a wheal. Cat, dog and rat fleas while preferring their normal host frequently bite man, and rat fleas in so doing may transmit bubonic plague. Fleas breed in cracks in floors and wallscots and unoccupied fleas lie in wait on the floor. Anti flea preparations should therefore be applied chiefly to the legs. Dimethyl phthalate sprayed on the trousers and socks or rubbed over the skin of the legs is the best preventative. Sprayed on the clothing

it is effective for several days. Other useful methods are the smearing on the skin of oil of pennyroyal oil of birch tar or oil of lavender or the dusting into the socks and underclothing of powdered pyrethrum, menthol or camphor. The irritation of a flea bite may be allayed by the application of 1:20 carbolic acid 10% menthol in spirit, or of one drop of carbolic acid and spirit equal parts or by rubbing in an antihistamine cream or an ointment containing carbolic acid 2% menthol 2% or liq. picis carbonis 12%.

The Bed bug (*Cimex lectularius*) appears to have been introduced into this country from the East only about 400 years ago. It is 5 mm. long 3 mm. broad and extremely flat. In colour it is rusty brown and it has an offensive smell. It lives in crevices in furniture and walls and may be transferred in luggage. The eggs hatch in a week or ten days, and the young bugs after five moults become adult in about twelve weeks. Bugs can live for over twelve months without food. The bite of the bug causes more inflammation than that of the flea. The irritation may be allayed by the application of any of the substances suggested for flea bites above. It is difficult to rid a building of bugs. fumigation with hydrocyanic acid gas is effective but dangerous. A modern method is to spray walls and furniture with a 5% solution of D.D.T. in kerosene. At least 100 mg. of D.D.T. per square foot should be deposited. This appears to render the surface lethal to bugs for three months (Buxton).

Harvest bugs. These are the six legged larval forms of various species of the acarid *Trombidium* and are normally parasitic on small mammals. They are bright red in colour and 0.3 mm. in length. They readily attack man in July August and September<sup>1</sup> if he walks through the grass or vegetation on which they are lying in wait, usually on chalky or light, sandy soils. The legs and ankles are the parts usually attacked. The mites attach themselves by their mouth parts to the skin, and

<sup>1</sup> I have been several bitten as late as 5th November when shooting, having supposed it to be late enough in the year to omit my usual prophylactic application of sulphur ointment.

inject a digestive fluid which causes a cylinder of coagulation or hyaline necrosis to form extending into the epidermis for a length equal to that of the mite. The mite sucks back the digested epidermis leaving a tube of hyaline material which is so solid that

it can be dissected out (Buxton). The bites of these mites cause a violent burning irritation which does not begin for a few hours but then increases for thirty-six hours. Protection is obtained by sprinkling flowers of sulphur or powdered naphthalene into the socks or underclothing from the knee downwards or by rubbing the legs with sulphur ointment. Dimethyl or dibutyl phthalate applied to the clothing or skin would probably be even better. The mites can be removed by mopping the skin with benzine or petrol, or taking a warm salt bath. The irritation

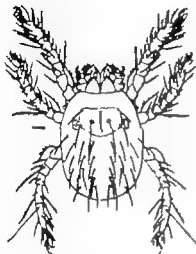


FIG. 52.

Harvest Bag *Microtrombidium*  
Arachnida.

(British Museum Economic Series, No. 2.  
Monthly Press. 1917.)

can be allayed by one of the preparations suggested for flea bites (p. 148). In endemic areas in S.E. Asia larval mites of this type are responsible for transmitting scrub typhus. In the campaign there impregnation of the clothing with dibutyl or dimethyl phthalate was very effective in killing these mites before they could bite.

**Ticks.** The common tick in this country is *Ixodes ricinus*, the wood or sheep-tick. It is dark brown in colour with a leathery integument. The female is 4 mm. long and 3 mm. wide when hungry and twice that size when full of blood. Ticks are normally parasitic upon vertebrate animals but are not very particular as to the species of their hosts. *Ixodes ricinus* for example attacks cattle dogs, sheep and humans impartially. Ticks are acari and have eight legs and a sharp probe or hypostome beneath the

mouth armed with recurved teeth. With this the tick penetrates the host's skin thereby attaching itself and sucking the blood. Every tick has three blood meals in its life viz. as a six legged larva as an eight-legged nymph and as an adult. Ticks, like harvest bugs, lie in wait for their hosts upon vegetation. A tick takes two to three days to get quite full of blood, after which it drops off. If it is forcibly removed before that time it is apt to leave its hypostome and the attached mouth parts *in situ* in the skin which will later give rise to local irritation and infection. If a drop of paraffin, petrol, turpentine, benzine, essential oil, or the tarry liquid out of a tobacco pipe is applied to the tick it will withdraw its proboscis after a few minutes or hours. The host is usually unaware at first of the attack of a tick but an urticarial wheal appears in a few hours and causes some irritation. This may be allayed by one of the preparations suggested for flea bites (p. 148). Diseases conveyed by ticks in different parts of the world are African tick fever due to *Spirochaeta Duttoni* and the various tick typhus fevers due to rickettsias, e.g. Rocky Mountain spotted fever. Indian, African and South American tick typhus also various diseases of animals.

**Bees wasps, hornets** The sting of these insects is always painful and may be dangerous. Danger may arise on account of the situation of the sting in the tongue or fauces, the resulting swelling obstructing respiration from the patient being anaphylactic to the venom or to the sting entering a superficial vein and the venom being injected directly into the blood stream.

The sting of these insects is complicated but its working is entirely reflex, so that a severe sting may be got from the abdomen of a bee which has been for many hours separated from its thorax. The poison consists of a mixture of formic acid and an organic base. The poison of the bee is acid while that of the wasp is neutral or alkaline (Speyer). Wasp venom contains large quantities of hyaluronidase and 2% of histamine (*Lancet* 1934 1 768). The poison sacs and glands are usually left by the bee attached to the sting. If therefore the sting is grasped with forceps more poison will be squeezed into the

wound. The sting should be removed by lifting or scraping it out with the edge of a knife. The usual application is solution of ammonia. This should be dabbed on not rubbed on or it will rapidly remove the epidermis. In the absence of ammonia a solution of sodium carbonate or bi-carbonate, or even a moist cake of soap, or bag of washing blue may be used.



FIG. 51.

Insect bites showing various degrees of reaction. Left forearm.

In the case of wasp stings, as the poison is neutral or alkaline dilute acetic acid vinegar or the juice of a lemon should be used. Hot fomentations are useful in severe stings. The treatment of cases where sudden collapse follows the sting should be on the usual lines for the treatment of shock and collapse.

Mosquitoes, midges and gnats. These insects may to some extent be kept away by smearing the skin with cod liver oil or with essential oils such as,

B	Ol cedar	5 iss.	III
	Ol citronellae	3 Hiss.	42
	Spt. camphorae ad	3 i., mince.	100

If a greasy preparation is preferred the following may be used,

R.	Ol. cedri	℥ 40	8
	Ol. citronellae	℥ 80	16
	Spt. camphorae	℥ 40	8
	Paraffin moll. alb. ad	℥ 1	100

A modern insect repellent is dimethyl phthalate, a colourless, practically odourless, oily liquid boiling at 230° C and less than 1% soluble in water. It should be rubbed over every part of the surface to be protected, avoiding the eyelids, lips and scrotum which are irritated by it. The protection lasts several hours if the stuff is applied to the skin and about a week if it is sprayed on the clothing. As it does not work by smell but by "burning the feet" of the insects which alight on it no exposed areas should be left untreated. It dissolves certain synthetic fabrics and plastics so care should be taken with artificial silk stockings and plastic spectacle frames. A 50% emulsion of it is strong enough to protect from midges.

The irritation from the bites of these insects may be allayed by application of 1-20 carbolic acid, 10% menthol in spirit or one of the other preparations mentioned under flea bites (p. 148). The bites of mosquitoes and especially of horseflies, are often highly septic and if a bite shows signs of being septic it is important that measures to combat the sepsis should be taken at the earliest possible moment either by constantly renewed boracic fomentations, hot saline arm or leg baths, Bier's treatment, sulphonamides or penicillin.

In certain patients the reaction to a mosquito bite may take the form of a large bulla possibly an inch in diameter. Such bullae may have to be diagnosed from those due to pemphigus vulgaris (p. 416).

## CHAPTER IX

### DISEASES DUE TO VEGETABLE PARASITES

#### SECTION I FUNGI

**Monilia.** The monilias (candidas) are yeast-like fungi consisting of ovoid spores some of which may show buds. They produce mycelium on the skin and in culture to a greater or less extent. *Candida albicans*, which can be separated from the others by its sugar reactions in culture, is the only important species which can become pathogenic. It is, however a normal harmless inhabitant of the gastro-intestinal tract.

**Thrush.** Thrush appears sometimes in the mouth of bottle fed babies as dry whitish patches on the tongue and buccal mucosa, composed of the fungus growing in the superficial layers of the mucous membrane. The patches are easily scraped off. The condition is also not uncommon in patients treated with the broad-spectrum antibiotics.

**TREATMENT** Careful sterilising of bottles and teats. Gentle mopping of the affected mucosa with mycostatin (p. 35) in a suspension containing 100 000 units per m.l., is rapidly effective, as well as being much less messy than the old treatment of 1% aqueous gentian violet. The thrush fungus sometimes causes an eruption in the napkin area (p. 119). The eruption consists of oval reddish macules tending to peel at the edges, the free edge of the peeling horny layer being directed towards the centre of the macule.

Monilia infection of the adult skin usually appears in the flexures, especially under conditions where two perspiring surfaces rub together e.g. axillae, groins, perinaeum, natal cleft (especially in patients being treated with the broad spectrum antibiotics) and under pendulous breasts. The lesions



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start as small red papulo-squamous patches which become vesicular. The vesicles rupture, exposing smooth red, moist areas each with an overhanging edge of white sodden epidermis



FIG 54.

Monilia infection of groin, perineum and anal cleft. Note overhanging edge of white, sodden epidermis.

in which the fungus can be found by examining scrapings in hypotannic under  $\frac{1}{2}$  in objective. This condition constitutes one variety of intertrigo the other being due to infection with streptococci and being a form of impetigo. Monilia may also infect the skin between and under all or most of the toes causing a moist, raw red and peeling condition. Also the skin of the

hands and feet, causing red areas with a fringe of epidermis at the margin or vesicular patches suggesting ringworm. It is also the cause of some cases of *paríche* (p. 188) and of the very chronic, cracked, white, leathery patches of skin which occasionally occur between certain of the fingers and are known as *erectio interdigitalis*.

TREATMENT of monilia infection of the skin. Mycostatin ointment and suspension (p. 35) have taken the place of the traditional 1% aqueous gentian violet or *pig magenta* (Castellani's



FIG. 53.

*Intertrigo under pendulous breasts.*

paint). They are both colourless and efficient. In intertriginous areas the suspension is better than the ointment. It should be painted on, allowed to dry and then powdered with zinc oxide or talc powder and the skin surfaces should be separated by a few layers of gauze. A 100r dose of X rays, repeated in a week, is a great help.

Chronic paronychia is another common form of monilia infection which affects women, usually housewives, who constantly have their hands in water. The nail folds of several of the finger nails become red, swollen and slightly painful. A similar appearance may be caused by infection with *B. coli* or staphylococci and streptococci of low virulence. The condition is very

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chronic and difficult to cure unless the hands are kept out of water. It is frequently associated with some degree of acrocyanosis or Raynaud's phenomenon.

**TREATMENT** Every effort must be made to keep the fingers dry by the wearing of fabric-lined rubber gloves. Medicaments are applied twice daily under the nail fold with a chisel pointed match or orange stick. Those cases entirely due to monilia respond well to mycostatin ointment. Great patience has to be shown in the treatment of the staphylococcal cases and the fol-

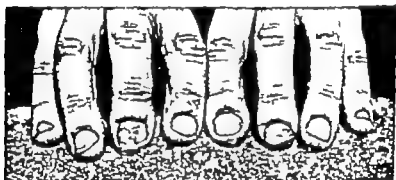


FIG. 56.

Chronic Paronychia, showing the eubiosis-like swelling of the nail folds.

lowing applications are all useful. 1 in 100 domiphen bromide (Bradasol) in 75% spirit, pig magenta (Castellani's paint) or 3% chlorhydroxyquinoline ointment (Vioform). All these measures are rendered more effective if combined with a course of X-rays, 75-100r weekly for three or four weeks. Sometimes it helps to seal the nail fold with colourless nail varnish, "Durofix" or "Portex" plastic skin.

Monilia may affect the nail plates themselves causing appearances rather similar to those produced by ringworm infections (p. 167) except that with monilia the lateral borders of the nails are chiefly affected and sometimes discoloured. This condition also responds to mycostatin ointment massaged in twice daily.

## RINGWORM

*Tinea* or ringworm is an infection of the skin hair or nails with a variety of fungi all of which have a segmented mycelium, the short segments being known as "spores" although they are not true spores botanically speaking. They are classified for clinical purposes as follows



FIG. 27

*Tinea circinata*. Back of Hand. *Ectothrix trichophyton*. Note postulation common in infections with this type of fungus.

*Microspora* or small-spored ringworms. Of these the commonest is *Microsporum audouinii* which causes almost all the cases of ringworm of the head in school children in the Midlands and the North of England and also attacks the glabrous skin, causing *tinea circinata*. Other *microspora* occur in cats, dogs, horses and guinea pigs and these frequently also infect man.

**Trichophyta** or large-spored ringworms. These are divided into the *Endothrix trichophyta*, in which the fungus is found only inside the fully infected hair. As it comes from the outside it can be found outside the hair also in the early stages of invasion. They are believed to be peculiar to man and to birds. *Ecto* or *Ecto endothrix trichophyta*, in which fungus is found

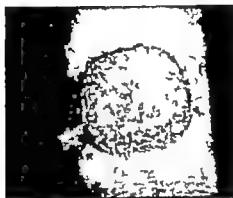


FIG. 58.

*Tinea circinata*. Front of upper arm.

both inside and outside the fully infected hair. These fungi normally attack animals (horses, cattle, pigs, cats, dogs, deer, etc.) and birds, but may be communicated to man in whom they are apt to cause very inflammatory lesions. There are two species classified as trichophyta on account of their microscopic cultural characteristics which are, however, incapable of attacking hair viz *T. interdigitale* and *T. rubrum*. *T. interdigitale* commonly causes vesicles on the feet. *T. rubrum* is the cause of very chronic infections of the feet and hands. Both are peculiar to man.

**Epidermophyta.** These do not attack the hair but only the horny layer of the skin or the nails. *Epidermophyton inguinale* (*E. floccosum*) is commonly the cause of ringworm between the toes and ringworm of the groins and axillae in this country. It is peculiar to man.

The diseases caused by ringworm fungi are highly contagious where conditions favour transference of the spores to a susceptible subject. The effects of the fungi on the skin are as follows. They cause the infected horny layer hair or nail to become brittle and friable, and they give rise to a more or less pronounced inflammatory reaction in the underlying corium. In some cases the toxins or even the spores of the fungus penetrate to the blood stream and are carried widely over the body giving rise to widespread eruptions known as microspoides, trichophyides or epidermophyides, according to the fungus concerned.

Tinea circinata, ringworm of the glabrous skin. This may be caused by either microspora, ecto- or endo-thrix trichophyta or by the epidermophyta. The lesions are more or less circular patches which spread out peripherally healing in the centre and so forming the rings from which the disease gets its name. They are red and scaly and, especially in the case of the ectothrix infections, may be vesicular or pustular and very inflammatory. The lesions are usually single or few in number but may be multiple. The incubation period is probably from 3-7 days. That of the very infectious small-spored ringworm to which blue Persian kittens are subject is 3-4 days (Whitfield). Incidentally ringworm in kittens cannot usually be diagnosed without the help of Wood's light (p. 44) as it often happens that only solitary hairs are infected, not patches recognisable by the naked eye.

DIAGNOSIS. Macular lesions which have not yet become rings have to be distinguished from *seborrhoea corporis* (p. 360) *psoriasis* (p. 339), *parapsoriasis* (p. 392)  *pityriasis rosea* (p. 355) and *discoïd eczema* (p. 323). Ringed lesions must be diagnosed from *seborrhoea corporis*, *psoriasis*, *pityriasis rosea*, the circinate forms of *impetigo* (Fig. 76) a form of *discoïd eczema* in which the centres of the patches have healed (p. 323) and in some cases from *tertiary syphilis* (p. 364). In all cases an absolute diagnosis can be made only by finding the fungus. To do this some scales should be scraped off from the periphery of the lesions or if vesicular the roofs of some of the vesicles should be cut off. These should be laid on a slide, covered with

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a cover slip and liq potassae run underneath the latter. The slide should then be warmed over a flame for a few minutes. The liq potassae should not be allowed to boil or the pieces of epidermis may be forced out from under the cover slip. If the slide is examined under  $\frac{1}{2}$  in. objective with a small stop the outlines of the epidermic cells will be seen forming a pattern like wire netting. If fungus is present it will be seen as wavy

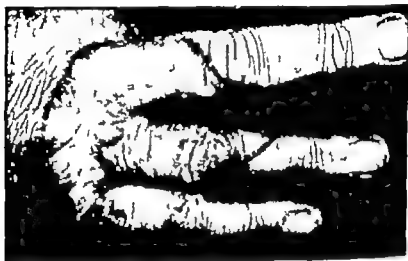


FIG 59.  
*Tinea circinata.*

branching lines cutting across the outlines of the cells (Fig 61). The mycelium of ringworm fungus usually shows a somewhat bluish tint at a certain focus and granules and vacuoles may be made out in the substance of it. The mycelium is usually divided at intervals by transverse septa. Any structure resembling mycelium which follows the outlines of the cells should be regarded with the gravest suspicion as being that form of artefact known as ghost or mosaic fungus." What this really is is not at present settled but for our purpose it should be regarded as an artefact produced by the liq potassae

Fungus is not always easy to find and if the pieces of epidermis are thick they should be boiled in liq. potassae in a test tube for five minutes. The liq. potassae should then be poured out into a Petri dish standing on a dark surface e.g. black paper and the pieces of skin fished out with a platinum loop laid on a slide and covered with a coverslip. Sometimes if no fungus is visible at first it becomes so after standing for twenty-four hours, the liq. potassae having in the meantime dissolved the epidermic cells.

**TREATMENT** There is a large number of fungicides available. It is doubtful if any one is more effective than the others, but they



FIG. 80.

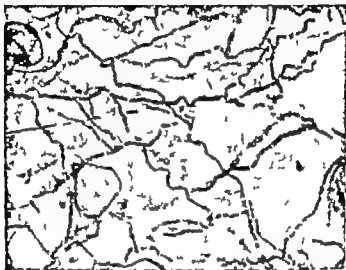
*Tinea circinata*, multiple lesions. Infection from cat.

all have one most important defect and that is that they do not penetrate nails or hairs. They are however effective for purely cutaneous infections, with the single exception of *T. rubrum* infections of the palms and soles. Examples are ung. acid benzoico (Whitfield's ointment), the formula of which is ac. salicyl 3%, ac. benzoico 6%, emulsifying ointment ad 100%. Ointments containing undecylenic acid (ung. zinc undeceno., Tinucifax), phenyl mercury complexes ("Mersagel", "Penotrans", "Tincol") and various other proprietary preparations. Finally in very resistant cases, dithranol 1% in p. molle may be used but caution must be exercised and a test application made.

Ointments are all best used on dry areas of skin. On moist areas, such as flexures and feet, lotions are better. These are obtainable containing any of the above substances or either per



magentae (*Castellani's* paint) or a modification of Whitfield's formula (ac. salicylic 3%, ac. benzoic 6%, acetone 50%, aqua ad 100%) may be used. Whatever preparation is used it should be applied twice daily and continued for a week after all signs of the malady have disappeared.



Dr. Klever & Robt. Smith

FIG 61

*Epidermophyton inguinale* in Epidermis (Liquor potassae). 300.  
Not faint outlines of the epidermal cells with mycelium of fungus  
cutting across them.

**Tinea cruris (Dhobie itch)** This eruption is produced by the *Epidermophyton inguinale* (*E. floccosum*) which grows in the horny layer of the skin and does not attack the hairs. The disease begins as red maculo-papules which spread peripherally and finally form red patches several inches across. The edge of the patch is generally more inflamed than the older central portion, and may be vesicular or pustular. The irritation is intense. The site of this eruption is usually the upper inner thigh on one or both sides, about the area which is in contact with the scrotum. It very commonly extends backwards on the perineum and into the natal cleft about the anus. It may also affect the

axillae the skin under pendulous breasts, and occasionally forms patches resembling *tinea circinata* on the thighs or abdomen.

**DIAGNOSIS.** The conditions which may be confused with *tinea cruris* and which are nowadays much more commonly seen, are *seborrhoeic dermatitis* (p. 393), *intertrigo* (p. 154) and *flexural psoriasis* (p. 339). The fungus is easy to find and should be looked



FIG. 62.

*Tinea cruris.* Note that edge is most active part of lesion.

for as described on page 159. The clefts between the toes should be examined at the same time.

**TREATMENT.** The condition clears up in one to three weeks with one of the fungicides already mentioned (p. 161).

***Tinea pedis.*** This is the commonest type of fungus infection. It is spread by the transfer of infected skin fragments in bath rooms, changing-rooms, swimming baths, etc. Thus it is particularly common in institutions where athletic games are encouraged, such as the armed forces, schools and universities.

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and in certain industries where communal bathing occurs, such as coal mining. The three common causative fungi are *T. interdigitale*, *E. floccosum* and *T. rubrum*. All cause maceration and peeling between the toes. The first two also cause vesication in this area and, when the condition occurs on the soles, groups of deeply set blisters which vary in size from 1 to 10 m.m. or more in size. *T. rubrum*, on the other hand, causes a dry scaling erythema on the sole. This infection is usually unilateral to begin with and may remain so for months or even years. The nails are also generally involved.



FIG. 62.

*Tinea pedis*. Involvement of toe cleft.

**DIAGNOSIS** The presence of sordid white skin, which comes away leaving raw areas, is extremely common between the fourth and fifth toes and is frequently due to the wearing of thick socks and tight shoes alone. When it occurs in other toe clefts as well, it is usually due to the presence of fungus. A diffuse eruption of vesicles between the toes and on the soles (podo-pompholyx, p. 336) is a form of eczema. The vesicles are not grouped and the

condition is usually bilateral from the start. Chronic eczema or dermatitis and psoriasis can resemble the eruption caused by *T. rubrum*, but the history and signs of the disease elsewhere usually allow a correct diagnosis to be made.



FIG. 64.

*Tinea pedis.* Involvement of sole

**PROGNOSIS.** *Tinea pedis* frequently relapses, some patients getting attacks year after year in the hot weather. The type caused by *T. rubrum* is extremely resistant to all forms of treatment.

**TREATMENT.** This is as described for *tinea circinata*. On the whole lotions are more efficacious than ointments. They should be painted on with a brush twice daily, allowed to dry and powdered with a fungicidal powder ("Asterol" "Mycil" zinc undecyloate etc.). Treatment should be continued for a fortnight after all signs of the disease have disappeared and the powder should be used indefinitely in cases which show a tendency to relapse. In some cases the condition may be severe and secondarily infected with pyogenic organisms. They should be put to bed, the feet should be bathed for 5 minutes twice a day in 1 : 8000 aqueous potassium permanganate, gently dabbed dry and then painted and powdered in the usual way. Systemic antibiotics may be needed to combat the secondary infection.

Prophylactic measures include the wearing of loose shoes with leather soles, cotton socks and the regular use of a dusting powder.

The condition caused by *T. rubrum* must be treated regularly if only to prevent spread to other individuals. In some cases eventual cure results. The two best applications are dithranol ointment or salicyl and benzoic acid lotion (p. 161).



FIG. 65

*Tinea unguium*, Ringworm of nails.

X Rays are useful in chronic cases, e.g. 200r into the affected interdigital cleft once a fortnight up to four times. Socks may be disinfected by boiling or by immersion for one hour in a 1% solution of thymol in indust. spirit (Kadiach) or by shutting them up in formalin vapour for 6 hours (Berberan). Shoes by shutting them up for 48 hours in a closed tin box containing 50 c.c of commercial formalin in a small dish (Henderson) or by swabbing out the insoles with 2% formalin (Gray).

**Tinea of the hands.** Only *T. rubrum* causes a direct fungus infection of the hand and this again takes the form of a dry scaly erythema. The condition often has a well defined margin and slowly spreads proximally up the fingers and hand over the years. It is usually unilateral and the nails, either on the fingers or toes, are generally involved. Treatment is the same as that described for the feet.

Widespread vesicular eruptions of the hands can accompany severe tinea pedis of any type but they are a secondary sensitization manifestation and fungus is found only in the lesions on the feet.

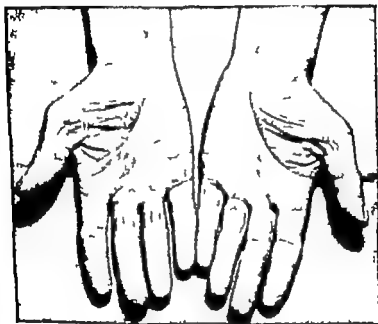


FIG. 68.

*Trichophyton rubrum* infection of right hand, left hand unaffected.

**Tinea unguium.** Ringworm of the nails. This is usually caused by *T. rubrum*, *T. interdigitale* or *E. floccosum*. The fungus usually gets under the free edge of the nail and grows back in the nail bed, eventually attacking the nail itself. Infection may less commonly begin at the nail fold or the lateral borders. One, several or all the nails of one or both hands or feet may be attacked. The affected nail is rough, opaque and friable and has an accumulation of scales under it.

**DIAGNOSIS.** Diagnosis can only be made with certainty by finding the fungus in shavings from the nails. These should

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be boiled in liq. potassae for five minutes or left to soak in it for twenty four hours, and the examination must be repeated several times before confidence can be felt in a negative result. The diseases liable to be confused with ringworm of the nails are *eczema*, *syphilis* and especially *psoriasis* (p. 345).

TREATMENT is exceedingly unsatisfactory. Probably the best method is to keep the nail constantly dressed with 6% salicylic and benzoic acid ointment under a covering of strapping. The strapping is removed every few days and the softened nail scraped away. Or a 12% solution of salicylic and benzoic acid in equal parts of indus. spirit and acetone may be painted on the nail twice daily the skin being protected with petroleum jelly.

I have seen several nails in several different patients apparently cured by the use of Thorium X, although in one or two patients not all the affected nails appeared curable in this way. The thorium should be used in a strength of 2000 c.a.u. in 1 c.c. alcohol and painted freely on the nail and run in under it for two days in succession in each week. Unless many nails have to be treated, the 1 c.c. of alcohol supplied can hardly all be used at one sitting. If the remainder is applied on the second day it will still retain about 80% of its strength. After the second painting the nail should be varnished over with collodion to retain the Thorium X as long as possible. The weekly paintings are continued until the nail has grown up healthy. If the skin about the nail gets sore or very red the treatment is suspended for a week or two to let it recover.

The usual result of removal of the nail by avulsion is that the new nail is infected in spite of all one's efforts to sterilise the nail bed. The only way I know of certainly curing ringworm of the nail which has resisted the treatments suggested above is complete surgical removal of the nail, nail bed and nail matrix.

*Tinea barbae* *Tinea sycosis*. Ringworm of the beard. There are two principal types. (1) The dry scaly type in which circular or ringed reddish, scaly lesions are covered with broken stumps of infected hairs. These look lighter in colour and

less shiny than the normal hairs, or they may break off close to the surface and show only as blackish dots plugging the hair follicles. This variety is due to the *Endothrix trichophyta* which are almost confined to man. It is usually acquired at the barber's from a shaving brush infected by a previous customer. The incubation period is 4-5 days (Cranston Low)

(\*) The suppurative type. In this there are red inflammatory papules, papulo-pustules or nodules surrounding a number of hairs on the beard region. There may also be some exudation



FIG. 67

Dr. S. S. S. S.

*Tinea barbae*, *Endothrix trichophyton*. Note indurated nodules.

of serum and crusting. The nodules often grow very large and form indurated purplish lumps on the chin and under the jaw, which are very characteristic of the disease. They appear to be produced by the fungus getting out of the hair follicle into the corium and setting up an inflammatory reaction there. This type of ringworm seldom affects the upper lip. It is due to



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*Ectothrix trichophyta* of animal origin and is therefore most commonly seen in farmers and cattlemen.

**DIAGNOSIS** depends on finding the fungus in infected hairs by examining in liq potassae under  $\frac{1}{2}$  in. objective. The preparation should be warmed or allowed to stand for half an hour before examination. In the black-dot type the stumps may have to be removed by a needle or comedo expressor. In the suppurative type the differential diagnosis is from *staphylococcal sycosis* (p 193) *impetigo* (p 182) *pustular acne* (p 403) *iodide* p 121) or *bromide* (p 122) eruptions and *sycphilides* (p 253). *Staphylococcal sycosis* commonly affects the upper lip and never forms the purple lumps so characteristic of *tinea sycosis*. It may be difficult to find infected hairs in the suppurative type of ringworm as those most heavily infected often fall out as a result of the inflammation.

**TREATMENT** The suppurative type is treated with wet gauze compresses of 1 : 10 000 aqueous hydrarg. perchlor. or lead lotion. Systemic antibiotics are frequently needed to combat the secondary pyogenic infection. Most of the infected hairs will be shed owing to the severity of the inflammation, but thorough daily manual epilation of all loose hairs will hasten recovery. When pustulation and crust formation have ceased a fungicidal ointment should be used twice daily.

The dry scalp type is much less common. It yields to topical fungicides, but treatment may have to be continued for two or three months.

**Tinea tonsurans** Ringworm of the scalp. This is most common in children under fourteen, and in them is due in the great majority of cases to one of the microspora. Recent figures (J. T. Duncan) suggest that in the Midlands and the North of England *M. audouinii* a "human" parasite causes practically all the cases, while in the South from Hampshire to Cornwall, the animal parasites *M. lanosum* (canis) and *M. felinum* (canis) take an equal or even greater share. In Portsmouth *M. lanosum* and in Devon *M. felinum* are almost exclusively the causes of ringworm of the scalp. In London the proportions of *M. audouinii* and *M. felinum* are about equal. (*Min. of Health Monthly*

*Bulletin* 1945 4 9<sup>o</sup>) Infections due to *T. Sulphureum*, an *Endothrix* parasitic to humans only are becoming increasingly common.

Ringworm of the scalp is rarely seen in adults, but when it occurs it is due to an *Ectothrix* or an *Endothrix trichophyton* or occasionally to an animal microsporum. Infections with trichophyta may also occur in children and in some countries are commoner than microsporum infections.



FIG. 68.

*Tinea tonsurans. Microsporum.*

**Microsporum infections.** *M. audouinii*. The disease is contracted by direct contact or by the medium of caps, hairbrushes, etc. the incubation period being 6-8 days. It appears first as a small greyish scaly patch covered with broken hairs. The patches increase in size and number and may involve the whole scalp. Patches of *tinea circinata* may appear on the glabrous skin at the same time.

The disease if untreated lasts until puberty except in cases

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where an acute inflammatory reaction ("Kerion") takes place. Then the patch becomes red, swollen and boggy and the hairs become surrounded with pus and fall out, so leading to spontaneous cure.

In cases due to animal type microspora, *M. lanosum*, *M. felineum*, (both=*M. canis*) the lesions tend to be more inflam-



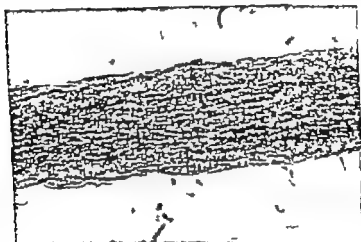
FIG. 80  
Kerion.

matory than those due to *M. audouinii* and are often curable by local applications without X-ray epilation. The fungus can be identified only by culture.

Owing to its infectious nature and the necessary quarantining of the patients ringworm of the scalp is of great practical impor-

tance, for it interferes seriously with the education of its victims. It is most important, therefore, for the practitioner to be able to recognise it.

The DIAGNOSIS must be made by the microscopic examination of suspected hairs, and it is therefore essential that the right



Dr. Kirby and Bold-Smidt

FIG. 70.

Scalp Hair infected with *Microsporum audouinii* (Liquor potassae).  
300.

hairs should be taken for examination. Hairs infected with *Microsporum audouinii* are usually broken off an  $\frac{1}{4}$  in. from the scalp. The stump which remains has a dull surface (due to its coating of spores) and usually looks lighter in colour than the healthy hairs. It has lost its elasticity (because its substance is broken up by mycelium) and consequently it is often bent at an angle and does not spring back to its normal position if rubbed "the wrong way". If such a stump is warmed in liq. potassae on a microscope slide and then examined with  $\frac{1}{4}$  in. objective it will be seen to be coated with a mosaic of polygonal spores all of one size (Fig. 70). These may be distinguished from fat droplets by the fact that the latter are perfectly round (unless deformed by pressure) and of various sizes (Fig. 1). Sometimes the coating of spores is rubbed off

the surface but they or the mycelial threads may be seen in the substance of the hair. A patch of small-spored ringworm should not be mistaken for one of *alopecia areata* for in the latter the skin is smooth not scaly the stumps are scanty not numerous, and the stumps themselves are shaped like exclamation marks (!) being very thin and depigmented at their insertion into the scalp while further up they possess their normal thickness, colour shiny surface and elasticity (p. 379).

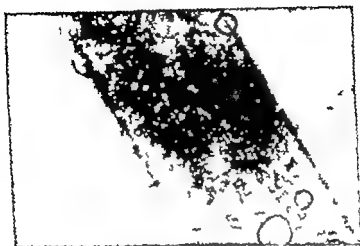
Scaly patches due to *psoriasis* and *dandruff* do not show the characteristic ringworm stumps.

A great help in diagnosis is provided by examination under Wood's light in a darkened room, for hairs infected with small-spored ringworm shine with a bright greenish fluorescence (p. 43). B. M. Partridge (*Trans St John's Hosp Derm. Soc.*, 1952, 31-34) has drawn attention to the importance in this disease of symptomless carriers with only a few isolated ringworm infected hairs which are not broken off and can be detected only by Wood's light.

**Endothrix infections.** The lesions are not scaly and the hairs do not have the appearances described above nor do they fluoresce under Wood's light. Often the infected hairs break off level with the scalp leaving the stump in the follicle, and causing "black-dot ringworm". In other cases the hairs may be  $\frac{1}{2}$  in. long or more. In liq. potassae the chains of large spores are seen inside the shaft of the hair (Fig. 72). To get out the stumps in black-dot ringworm a needle or a comedo expressor may be necessary. Endothrix cases may be mistaken for mild, patchy seborrhoeic dermatitis or even *alopecia areata*.

**Ectothrix infections.** These are derived from animals and are usually much more inflammatory than either of the preceding types, and consequently have a tendency to spontaneous cure. In liq. potassae chains of large spores are seen both inside and outside the hair. There is no fluorescence under Wood's light.

**TREATMENT OF SCALP RINGWORM.** The first step is to clip the hair as short as possible all over the head with clippers. This



Dr. Kleber and Roth-Schell.

FIG. 71.

Fat droplets on a normal hair (Liquor potassae).  $\times 300$ .  
Note bristly cuticle of hair and very variable size of the fat droplets.  
For comparison with Figs 70 and 72.



Dr. Kleber and Roth-Schell.

FIG. 72.

Fungal Hair infected with an *Endothrix trichophyton* (Liquor potassae). 300.

will often show that the disease is much more extensive than was supposed. In the suppurative cases bathing the head twice daily with hot water and the application of a 3%-6% benzoic and salicylic acid ointment or one of resorcin 2% will probably be all that is required.  $\lambda$  rays should not be used. In non-suppurative cases the hair of the whole head must be removed by X rays if there is to be any hope of cure in a reasonable time. An exception may be made in cases proved by culture to be due to animal type microspora which seem often to be curable by local treatment only (Thomas, Lennox & Duncan, *B.M.J.* 1945 2 316). In these cases the various methods of treatment given for *trichophyton circunata* (p 161) may be applied, remembering that dithranol if it gets into the eyes, will set up an acute conjunctivitis.

By the use of penetrating ointment bases containing salicylanilide or phenylmercuric nitrate and meticulous care in treatment Brain and Haber (R T Brain et al *B.M.J.* 1948 1 723) (H Haber et al *B.M.J.* 1949 2 626) following American work, were able to cure about 75% of their cases of ringworm of the scalp without the use of  $\lambda$  rays in an average of 2½ months. No difference was noticed in the results between *M. audouini* and *M. felineum* infections but the children of certain families appeared more difficult to cure than others. The resistant 25% were cured after X ray treatment. The ointment base used was as follows,

1 Crill No 6	10 c.c.
Solution of Citric Acid 3% and )	2 c.c.
Sodium propionate 1	
1 Carbowax 1500 to	100 gm.

Melt the carbowax 1500 over a water bath add the Crill No. 6, dissolve the salicylanilide in the mixture, add the solution of citric acid and sodium propionate and allow to cool, stirring occasionally.

In one series of cases 0.5% of phenylmercuric nitrate and in another 5% of salicylanilide was used as the fungicide incorporated in the above base. There was little to choose between

the results. In both series the hair was kept clipped short, the head was scrubbed gently with soap hot water and a soft nail brush night and morning, and the ointment rubbed in twice or three times daily with a tooth brush all over the scalp. A linen or cotton skull cap was worn all the rest of the time and the children were examined every three weeks by Wood's light. These good results, however probably depended on the care and attention given to the whole regime of treatment and not to the so-called penetrating bases.

**X RAY TREATMENT** The head is given 400r of X rays to five areas—frontal, vertical, and occipital and one over each ear (Adamsen-Kienböck) or to four areas omitting the vertical (Schrens). The intervening areas receive a partial dose from two or more of the areas directly treated, and the net result is that the whole hairy scalp receives an approximately even dose of 400r. For details vide Shanks, *Brit Jour Derm & Syph.* 1931 43 477 and 1932, 50 440.

About the eighteenth day following the treatment the hair becomes loose and falls out during the next week. At the end of four weeks the head should be perfectly bald. If some hairs are left it does not matter provided all the infected hairs have been removed. During the period from diagnosis to complete epilation the head must be washed every night and kept greasy with a 3%-5% benzoin and salicylic acid ointment made with an emulsifying base not only to kill any exposed fungus, but to prevent loose hairs from falling about and infecting other children. Skull caps of cotton which are boiled on alternate days should be worn during this period. Children down to one year can be X rayed, but under the age of four suitable restraining apparatus is usually necessary to keep them still. If this is not available full doses of chloral and Pot. bromide for three or four days preceding the treatment will usually calm a restless or nervous child sufficiently to make X-ray treatment possible.

If a few ringworm hairs are left they can be removed by cautious painting of the affected area with croton oil on two or three occasions. This causes a purulent folliculitis, loosening the hairs, which can then be pulled out with forceps under Wood's light.



If a large area of infected scalp has failed to epilate it must be X rayed again, but not for three months after the first X rays, lest permanent alopecia should result.

Parents often ask that only the infected area should be X rayed. I have occasionally done this and got away with it, but in many cases it fails and other ringworm patches appear. The patch already X rayed then interferes with the proper epilation of the rest of the head. Partial epilation is only justifiable if there is only one patch as proved by clipping the entire scalp and examining by Wood's light. The head must then be treated by daily washing and constant application of 3%-6% benzole and salicylic acid ointment for two or three weeks. If, on further clipping and re-examination, no more patches are found to have developed then X raying of the one affected patch and a margin of an inch all round it may be tried, but I do not recommend it.

When every ringworm hair has fallen out and none can be detected by Wood's light, the child is cured and may return to school. The hair will not begin to grow again for two months, and the child will not have a good head of hair for five to six months. After X ray epilation hair previously straight often returns curly but the change is unfortunately not permanent.

**CONTRA INDICATIONS TO X RAY TREATMENT** A head with a pustular or impetiginous infection should not be X rayed until the pus infection is cured otherwise it will flare up and may spread all over the scalp. If the scalp has been irritated, e.g. with strong iodine it must not be X rayed until all evidence of irritation has subsided. Suppurative ringworm (Kerion) should not be X rayed.

**DANGERS OF X RAY TREATMENT** The only danger of importance is the slight risk of over-exposure resulting in permanent epilation and some damage to the skin. For this reason X-ray epilation should only be undertaken by those who have acquired much skill and experience in this type of work.

**Trichophytides etc.** Distant eruptions due to local ringworm infections are called trichophytides, microsporides or epidermophytides. The term mycids includes these and similar

eruptions due to other fungi. A common one is the vesicular epidermophytide on the palms due to ringworm of the toes (p. 107)



FIG. 2.

Trichophytide, Lichenoid, follicular type.

Deep-seated inflammatory ringworms occasionally gave rise to generalised eruptions on the trunk and limbs. The commonest is the lichenoid trichophytide or lichen trichophyticus,

an eruption of small red follicular papules sometimes acuminate and resembling lichen spinulosus. Vesicular and pustular lesions occur less frequently. Lesions resembling erythema multiforme have also been described. These generalised eruptions may apparently be due either to sensitisation to the toxin of the ringworm fungus, or to actual dissemination of the spores of the fungus by the blood stream and their deposition in the skin. They get well when the original infection is cured.

**Other fungi infecting the skin** *Trychophyton schoëlemi* (man) and *quinckeanum* (mouse) are the causes of favus. *Microsporon minutissimum* (*Nocardia minutissima*) is the fungus causing erythrasma. *Microsporon* (*malassezia*) *furfur* is that causing pityriasis versicolor.

Favus is not a common disease in this country although it is endemic in Edinburgh, Dundee and Dublin. In London it is usually seen only in immigrants from Eastern Europe, except for occasional cases of mouse favus. It is characterised by sulphur yellow cups 2-3 millimetres in diameter embedded in the skin, each one surrounding a hair. These cups (scutula) are composed of a dense felt-work of mycelium. Quite commonly the characteristic scutula cannot be found and diagnosis will depend on careful microscopic examination and culture of hairs. Suspicion should be aroused in any case of chronic, patchy cicatricial alopecia with some crusting and scaling, especially if the scalp has a mousey odour. Favus may persist into adult life, causes permanent patchy cicatricial alopecia, and is much more difficult to cure than ringworm although the treatment is on the same lines. Favus may also attack the nails.

*Pityriasis versicolor* (*Tinea versicolor*) appears as café-au-lait coloured patches or sheets, usually on the upper part of the trunk. The affected skin may show slight wrinkling and scales readily on light scratching. Usually there are no symptoms. The disease is most common in those who sweat freely. It may be mistaken for the pigmentation due to arsenic (p. 119) or Addison's disease, or the white patches of normal skin may be mistaken for white areas of vitiligo but if the scales are examined in liq. potassae the fungus, *Malassezia furfur* is easily

seen as fine threads of mycelium in short lengths with masses of small round spores. The disease is due to *Malassezia furfur* (allied to the monilias) growing in the cells of the horny layer. It produces no inflammatory reaction and therefore has no tendency to spontaneous cure.



FIG. 74.

*Pityriasis versicolor*

**TREATMENT** : Frequent washing, frequent changes of under clothing which should be of cotton or linen so that it can be boiled and the daily application of a 3% ointment of salicylic

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and benzoic acids, or of 2% salicylic acid and sulphur or a lotion of sodium thiosulphate  $\text{℥ i. ad } \text{℥ i. (12\%)}$

The disease is easily cured, but readily relapses unless the treatment is persisted in for a week or two after apparent cure.

**Erythrasma.** A chronic infection of the horny layer cells of the inner upper thighs adjoining the scrotum with *Nocardia minutissima*. Sometimes the axillae are affected. The disease appears as a brownish scaly or wrinkled area of a uniform surface, i.e. the disease is no more pronounced at the edge than in the centre of the patch. It is of very slow growth and causes no symptoms, except occasional irritation when the patient perspires. Patients therefore are often unaware of its presence.

**DIAGNOSIS.** From *Tinea cruris* This is of rapid development, is decidedly inflammatory the edge is more inflamed than the centre of the patch and irritation is very pronounced. From *intertrigo* Here the skin is sodden and moist the surface is often eroded and inflamed and the edges are rather indefinite.

An absolute diagnosis can be made by finding *Nocardia minutissima* in the scales in liq. potassae. The mycelium is very fine and the spores very small. A  $\frac{1}{16}$  inch oil immersion lens is required to see this fungus properly.

**TREATMENT.** Benzoic and salicylic acid ointment, 3-5% in an emulsifying base.

## SECTION II STREPTOCOCCI AND STAPHYLOCOCCI

Impetigo contagiosa, "acrum pox" is an infection of the superficial layers of the epidermis, caused in about 90% of cases by pyogenic staphylococci the remainder being due to streptococcus pyogenes. Over three-quarters of the staphylococci are of a single type (type 71) the majority of which are penicillin resistant and an equal proportion of the streptococci belong to two closely related serological types. The malady is thus seen to be a fairly distinct infection and not one due to invasion by arbitrary organisms normally resident on the skin.

The features of the disease are the same whichever the causative organism. In either case a vesicle is formed. This ruptures

in an hour or two in some cases, but not for a day or two in others. In the former thick, yellow translucent, "stuck-on" crusts are formed from the exuding serum, but traces of the extending vesicle can usually be seen at the external edges of the crust. In the latter little or no crust is formed and the vesicles enlarge up to one to two inches in diameter sometimes healing completely in the centre and extending at the edges.

When the crusts are removed the serum continues to pour out and very soon reforms the yellow crust. The original lesion enlarges and new lesions soon appear in the neighbourhood of it. Picking off the crust with the fingers often leads to the formation of a purulent blister alongside the nail of one or other index finger.

The disease is very contagious both from one part of the body to another and from one individual to another. The slightest abrasion in a patient with impetigo is almost certain to become the site of a new lesion. The disease may readily be conveyed by towels, sponges, shaving brushes, etc., and is one of the diseases popularly known as barber's rash. Its incubation period is 2-3 days. It is commonest in children, but frequently occurs in adults. The commonest site is the face but in the presence of scratched lesions due to pediculosis capitis or corporis, scabies, lichen urticatus, midge bites, eczema or seborrhoeic dermatitis it may occur widely over the scalp or the whole body and limbs. It readily occurs on existing lesions of herpes simplex about the mouth, and of seborrhoeic dermatitis behind the ears. It may arise primarily on the auricle from a streptococcal middle-ear discharge.

On the scalp and beard the staphylococci soon invade the hair follicles and set up a purulent folliculitis (syccosis) which on the beard may be much more difficult to cure than the original impetigo. The lymph glands into which the site of the infection drains are often, but not necessarily enlarged.

COURSE. The disease has a tendency to clear up after persisting for about three weeks, though many cases last much longer and cases may often be cured much more quickly by skilful treatment.

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**Diagnosis** The ordinary case is easily diagnosed but the circinate type is sometimes mistaken for ringworm. In the latter the ring is nearly always made up of a number of minute



FIG. 75.  
*Impetigo contagiosa*

individual vesicles or pustules, while in impetigo it consists of the continuous edge of one large vesicle whose centre has disappeared.

A very acute impetigo involving practically the whole of both



FIG. 78

Impetigo contagiosa. Crustate type. Note extending edge of vesicle beyond the crust in many of the lesions.

sides of the face almost symmetrically may be extremely difficult to diagnose from an acute weeping eczema of the face. It is difficult to give any exact criteria by which diagnosis may be made but help may sometimes be obtained from the history—impetigo usually starting on one side and spreading, while



eczema usually starts symmetrically on both sides. The presence elsewhere of characteristic lesions of one or other disease would, of course, help in the diagnosis.

According to Twiston Davies many cases of supposed resistant impetigo are really examples of seborrhoeic dermatitis.

Bullous impetigo of the newborn (*pemphigus neonatorum pemphigoid*) is usually seen nowadays in maternity wards, and in all the recently reported outbreaks the infection has been by *staphylococcus aureus* in pure culture. The origin of the *staphylococcus* in these cases has been from ordinary septic lesions such as whitlows boils or impetigo occurring in the doctor nurse, or mother or in some cases in the laundry workers who folded and packed the clean clothing. In one series of cases the sterile cotton wool used in the ward was found to be heavily infected with *staphylococcus* (Hart 1938).

**DESCRIPTION** The age of onset is usually in the first few days of life, but may be up to three weeks, and the disease may also occur in older children. The disease occurs in two types, severe and slight. The severe cases tend to occur at the beginning of an epidemic. The disease starts with a small vesicle, sometimes preceded by a red or yellowish spot, on any part of the body except the palms and soles. This rapidly enlarges and new lesions appear around it. The vesicle contents are clear at first but become turbid in 12 to 48 hours. After 48 hours or so the vesicle ruptures leaving a raw surface with ragged edges. The size of the lesion is usually from  $\frac{1}{2}$  to 2 cms. in diameter but may be much larger. In the severe cases the disease extends rapidly and very large areas of the body may be denuded of epidermis. Such cases are usually fatal from staphylococcal septicaemia (via the umbilical cord) between the 4th and 12th day. They generally have a raised temperature but even fatal cases may be afebrile throughout. In ordinary cases the disease continues to extend for about two weeks, then the formation of fresh bullae ceases rather suddenly and all lesions heal in about one more week. Constitutional symptoms are mild or absent. Although as a rule the lesions are bullae which leave raw areas, crusted lesions like those of

ordinary impetigo are sometimes seen. The stages of the disease seem to be as follows

Incubation	1 to 3 days.
Spread	14 days.
Healing	- 7 days.

The duration of an ordinary case is about three weeks.

**PROGNOSIS.** The fatal cases usually occur at the beginning of an epidemic. Therefore if the epidemic is rapidly controlled the percentage mortality appears high. If the epidemic continues for a long time the majority of the cases are usually mild and the percentage mortality is low. Early and rapid extension of the bullae is an unfavourable sign. The disease leaves no scars.

**DIAGNOSIS.** The principal diseases from which diagnosis must be made are the bullous syphilide of the newborn and halogen eruptions. The bullous syphilide is more or less symmetrical, it is either present at birth or appears in the first three days after birth. In this the bullae are most numerous about the hands and feet, including the palms and soles which are never affected by bullous impetigo. The syphilitic child also will probably be wasted, have a harsh *cough* *au lait* coloured skin, snuffles, hoarse cry enlarged liver and spleen, and a positive Wassermann reaction. The child with bullous impetigo will probably be otherwise healthy and the lesions not symmetrical.

Halogen eruptions are sometimes bullous but they tend to be persistent and a history can usually be obtained of the administration of "soothing powders" to the baby or of bromide or iodide to the mother. Other bullous conditions are very rare in the newborn, and in all of them the vesicle contents would at first be sterile, while in bullous impetigo the content of the earliest vesicle gives a profuse growth of *staphylococcus aureus*.

**PROPHYLAXIS.** Overcrowded wards, imperfect asepsis, and allowing visitors to handle babies all increase the risk of an outbreak. Nurses should report the slightest septic lesion noticed either in themselves or in a mother or child. Periodic tests should be made of the efficacy of the sterilizing of dressings.

and towels. The supervision of the laundry is particularly important as epidemics have been traced to laundry workers. When the first case occurs it should be isolated and its nurse should nurse no other children. A careful search should be made for the source of the infection with a view to its elimination. Unhealed or recently healed whitlows, boils, and impetigo are the commonest causes, but staphylococcal nasal discharges and sterile wool containing staphylococci have been incriminated in different outbreaks. It must be remembered that a patient with a septic lesion anywhere is likely to have pyogenic cocci widely distributed over his skin surface.

Bullous impetigo of adults is common in the tropics and occasionally occurs in this country.

**Ecthyma** Sometimes the infection in impetigo penetrates deeper than the epidermis and involves the dermis. Ulceration then takes place, and the crusts are of a dirty brown colour and have pus underneath them. Such lesions are called ecthyma, and, because of the involvement of the dermis, take longer to heal than ordinary impetigo and leave permanent scars.

**Intertrigo** One form of intertrigo is due to monilia (p. 151) another is caused by the streptococcus pyogenes and is really impetigo affecting a moist flexure. It may occur in the groins, perinaeum, natal cleft, axillae and under pendulous breasts. The skin of the flexure is raw and red, with some sodden epidermis upon it, and at the border of the lesion the remains of the characteristic vesicle edge can be made out.

**Scaly impetigo (Pityriasis simplex)** Dry scaly patches with the scales arranged more or less in parallel rows, like a ploughed field, are often seen on children's faces. These may occur where a patch of ordinary impetigo has dried up or they may arise *de novo*. They are made worse by alkaline or carbolic soaps and by east winds. They are believed to be a chronic form of impetigo and are sometimes very resistant to treatment.

**Perleche** is a form of scaly impetigo which occurs round the mouth and is associated with cracks in the lips and at the angles of the mouth. It is aggravated and probably originated

by licking the lips. A similar condition can apparently be produced by infection with monilia.

**TREATMENT OF IMPETIGO** The average case of impetigo can be cured in 5-7 days with a locally applied antibiotic. Heavy crust



FIG. 77

*Pyriame simplex*, three months duration. Patient used carbolic soap.

ing should be gently removed by bathing with warm water and  $\frac{1}{4}$  . neomycin ointment or lotion or 1% tetracycline or chloramphenicol ointment gently smeared on thrice daily. In bullous cases the roofs of the blisters should first be cut away and in widespread cases especially in infants, systemic antibiotics may be necessary.

*Ecthyma* should be treated on the same lines as ordinary impetigo.

*Streptococcal intertrigo* should be treated on the same lines as *seborrhoeic intertrigo* (p. 368).

the occasional mild superficial follicular pustules from which *everyone suffers at times*, while *Staphylococcus pyogenes aureus* is the cause of the more severe follicular inflammations known respectively as impetigo of Bockhart, sycosis, sycosis nuchae or acne cheloid, boils and carbuncles. Boils are produced when the inflammation is at first situated at the bottom of the hair follicle where necrosis and pus formation take place the pus only later reaching the surface. Carbuncles are caused when the necrosis extends beneath the surface and forms a horizontal plate involving many hair follicles together with the



Dr. Adanson's Coll.

FIG. 79

Impetigo of Bockhart. Forcema.

intervening tissue over an area which may be several inches across.

**Impetigo of Bockhart.** This is a superficial pustular folliculitis which may occur on any hairy area. It is sometimes started by the application of some irritating ointment, such as one containing mercury or tar or by rubbing with camphorated oil. At other times it arises in the neighbourhood of a septic wound. A common site is on the front of the thighs in a hairy man. There is first a red papule at each follicle. This rapidly develops into a superficial yellow pustule surrounding the hair. Owing to transference of staphylococci to adjacent follicles the

disease spreads easily and when once established is sometimes resistant to treatment.

**TREATMENT** The pustules should be opened and the whole area treated with a local antibiotic such as 0.5% neomycin lotion. Alternately 1% brilliant green or gentian violet in 75% spirit or 0.5% quinine emulsion (p. 195) may be used. In severe cases, systemic antibiotics may also be necessary.



FIG. 62.

Dr. Johnson Case

Sycosis barbae. Cosmograph.

**Sycosis** Sycosis is an infection of the superficial parts of the hair follicles of the beard region with *Staphylococcus aureus*. It sometimes arises as such, but very often supervenes on an attack of impetigo contagiosa. As it is often very difficult to cure, it is important that every attack of impetigo on the beard region of a man should be taken seriously and cured as quickly as possible lest sycosis supervene. Sycosis is one of the conditions included in the popular term "barber's rash" as it is often acquired at the barber's. The incubation period is at least 48 hours (Sequeira). Owing to the inflammation extending deeply into

the hair follicle and outwards into the surrounding corium there is often considerable inflammation of the affected skin as a whole and the condition is apt to become chronic and to last for years. The commonest sites are the upper lip and centre of the chin, but the disease frequently involves the whole of the



FIG. 81

*Syphilis Barbae*. This type has to be diagnosed from *Framboesiform syphilide* in the same situation.

beard region. When it occurs on the upper lip it is often the result of a chronic rhinitis the hair follicles being infected by the nasal discharge. In many cases there is an associated *ptyriasis capitis* and a tendency to "seborrhoeic" eruptions. Ingram (*Brit. Jour. Derm. & Syph.* 1938 50 119) has drawn attention to the frequency of antral and other sinus infections in chronic

sycoosis. The nostrils often harbour virulent staphylococci without any local lesion.

The characteristic picture is at first a group of red follicular papules. These rapidly become converted into yellow pustules, each surrounding a hair. The intervening skin becomes red and inflamed. Shaving aggravates the process and spreads the infection.

**DIAGNOSIS.** From *Ringworm of the beard* (p. 168). This seldom attacks the upper lip and the common form of it is characterised by the presence of purplish inflammatory nodules which are absent in staphylococcal sycoosis. Even if there are no nodules the infected hairs will be brittle and will show ringworm fungus under the microscope. In staphylococcal sycoosis the strength and texture of the hairs are unaffected.

From *Lupus vulgaris* (p. 206). By the long history of this disease, the presence of apple-jelly nodules and absence of pustules surrounding the hairs.

From the *Framboesiform syphilids*. This often occurs as an infiltrated papillomatous patch in the middle of the chin, an area favoured by sycoosis. The syphilids however may be present also at the angles of the mouth, the sides of the nose, and on the scalp. Other signs of syphilis, including a positive Wassermann, will probably be found if looked for.

**TREATMENT OF SYCOSES.** Mild and recent infections usually yield rapidly to local antiseptics, though relapses are common. Very rarely in intractable cases, systemic antibiotics may clear a case when all else has failed. The second most important local applications are the quinoline derivatives, each as 0.5% quinoline emulsion (benzoyl peroxide 10 pot hydroxyquinoline sulphate 0.5 H.L.B. simplex 25 water to 100), or one of the proprietary preparations (Quinolol ointment, Vioform cream, Steroxin cream). The patient should stop shaving but should cut the beard as short as possible with scissors. Manual epilation of infected follicles is sometimes helpful. Finally small doses of X-rays are helpful, although X-ray epilation is no longer practised.

In spite of these measures, cases are still met which are



the external auditory meatus where the tissues are tightly bound down, they are extremely painful. Boils about the nose and upper lip are sometimes fatal owing to the infection reaching the meninges via the angular vein. A "blind boil" is one which is slow in pointing, and may exist for weeks. A threatened boil sometimes subsides and disappears in a few days without pointing.

**HISTOPATHOLOGY** The picture depends upon the stage of the infection. There is acute inflammation of the deeper parts of the hair follicle with thrombosis of small vessels, necrosis and dense infiltration with leucocytes and small round cells in the corium and subcutaneous tissue.

**TREATMENT General.** An examination of the patient should be made to exclude diabetes, nephritis, scabies or other disease. An open-air holiday is useful if the patient is run-down. The diet should be varied and should contain plenty of green vegetables, carbohydrates should be limited and sugar should be excluded. The bowels must be kept open. Systemic antibiotics are sometimes necessary and on occasions a five day course of tetracycline may even cure a case of recurrent boils. Vaccines rarely if ever have any beneficial effect. Injections of staphylococcal toxoid used in a carefully controlled series of cases from my department by Drs. R. Hlaver and A. Q. Wells proved to be valueless. Yeast and its preparations are often prescribed but I have seldom seen any effect from them. By far the most successful general treatment that I know is a course of 8-12 ultra violet light baths to the whole body given two or three times a week. In my experience this seldom fails to stop a crop of boils.

**LOCAL TREATMENT Abortion.** A threatened boil may sometimes be aborted by a single dose of X rays (100 or 200r) by the continuous application of a mercury and carbolic plaster sometimes by the extraction of the hair from the follicle sometimes by painting with iodine repeatedly.

The best treatment for a boil is probably as follows. Administer if possible a dose of X rays (100 or 200r) to the boil or give a brisk erythema dose of ultra violet light to the boil.

and its surroundings. Paint the area around the boil with a 1% solution of crystal violet, brilliant green or antibiotic lotion. Then cut out a disc of white chiroplast or felt with adhesive back,  $\frac{1}{2}$  inch thick, two to three inches across, and with a central hole big enough to fit easily round the boil and stick it firmly in position. Partly fill the central hole over the boil with a loose pad of cotton wool moistened with glycerine or 12% sodium sulphate solution and secured by a narrow strip of strapping across the hole. This pad may be renewed night and morning. The felt remains in situ till the boil is cured. This device isolates the boil and so prevents others arising, keeps pressure off it and keeps it at rest while the glycerine or sodium sulphate tends to promote drainage once the boil has pointed. When the boil has softened in the centre, but not before, it should be opened with a galvano-cautery or with a pointed match-stick dipped in phenol. Either of these can be used without removing the felt ring. If the felt ring device is not used a ring of contractile collodion painted round the boil hastens its resolution. As a general rule fomentations should not be used on boils as they tend to spread the infection. A 1% crystal violet or brilliant green or antibiotic lotion should be painted on the skin round a boil to prevent others arising.

Boils in the external auditory meatus should be treated by hot bottles or radiant heat to the outside and by mopping out the meatus frequently with carbolic acid in glycerine, e.g. glycerine of carbolic acid 3 fl. glycerine to 5 l. (i.e. 4% of phenol).

Boils on the upper lip and nose should be bathed frequently with hot water or covered with magnesium sulphate and glycerine paste and when pointing should be opened with a cautery not with a knife, so as to minimise the risk of opening up fresh vascular channels for infection. For the same reason the patient must be cautioned seriously about the danger of squeezing boils in this situation as many fatalities have been recorded.

Small doses of X rays, e.g. 50-100r repeated weekly three or four times, often clear up an area of constantly recurring

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boils promote healing of boils which have pointed, and prevent the formation of indurated scars.

Boils in the axillae can be exceedingly troublesome owing to the fact that the large apocrine sweat glands open into the hair follicles and so become infected. The hair must be removed by a depilatory shaving, or clipping as short as possible with scissors. A 100r dose of X rays if available should be given to the whole axilla. The axilla should be painted once or twice daily with a 1% solution of crystal violet or brilliant green or antiseptic lotion to try and prevent any new boils arising. Heat should be applied by an electric light bulb or hot bottle and dressings of magnesium sulphate and glycerine paste, or gauze wetted with glycerine or with 12% sodium sulphate solution applied. Boils which soften should be opened with a galvano-cautery. The X rays should be repeated weekly three or four times and it may even be necessary to epilate the axilla with X rays. As staphylococci are widespread on the skin in cases of boils the patient should sponge himself down with 1% cetrimide after his bath.

**Carbuncles** A carbuncle may be regarded as a mass of confluent boils. The affected hair follicles, and the intervening tissue in the deeper part of the corium form a continuous slough bathed in pus, and surrounded by a dense phlegmonous inflammation of the subcutaneous tissue and skin. Carbuncles may be several inches in diameter and are usually single. A common site is the nape of the neck. They are most common in men past middle age, while diabetes and alcoholism are predisposing causes. They often cause considerable constitutional disturbance.

A carbuncle begins as a flat, dusky red, painful, infiltrated area. This extends steadily for 7-10 days when it softens at numerous points on its surface. These perforate and liberate pus. The perforated skin subsequently gives way and the large central slough eventually separates and is discharged the cavity healing by granulation and leaving a large scar.

The prognosis in a healthy patient is good although the healing may take some weeks. In elderly or debilitated patients, diabetics and chronic alcoholics, a carbuncle is not rarely fatal.

**TREATMENT** *Local.* Conservative treatment consists in local and general rest with X rays 50-100r repeated in three to seven days respectively up to a total of about 300r. Magnesium sulphate paste or kaolin poultices externally the skin about the carbuncle being painted twice daily with 1% brilliant green or antibiotic lotion. Puncture of soft spots with a cautery and replacing the pus with penicillin solution or if this is not available, applying wet dressings of 12% sodium sulphate to help drainage. Surgical treatment consists in complete excision and suture or thorough scraping under a general anaesthetic followed by spraying the cavity with penicillin solution four-hourly or by packing it with magnesium sulphate and glycerine paste, which is renewed once or twice a day. Skin grafting may accelerate healing when granulation is established.

*General.* Systemic antibiotics are usually necessary. Good food, fresh air and general ultra violet light baths help to keep up the patient's resistance.

**Granuloma pyogenicum.** These are small, dark red or purple, tumours,  $\frac{1}{4}$ - $\frac{1}{2}$  in. in diameter often pedunculated, which consist of vascular granulation tissue covered by a thin epidermis. They arise on the sites of small septic wounds, of which there may be no history and are caused by infection with staphylococcus pyogenes. They grow very slowly and may persist indefinitely. They have to be diagnosed from vascular naevi (p. 57).

**TREATMENT** Cutting off with the galvano-cautery if pedunculated or destruction with the same if sessile is usually successful. If the lesion returns x-ray ionisation is reliable.

**Marginal blepharitis.** An exceedingly chronic staphylococcal infection of the eyelash follicles common in patients with dandruff (p. 388) syrosis (p. 103) or rosacea (p. 409). The edges of the eyelids are inflamed and tend to stick together in the morning. Small pustules may occur around some of the lashes. The general health and the accompanying dandruff, syrosis, etc., must be treated. Locally antibiotics, especially when combined with hydrocortisone for the subacute and chronic cases, are the most effective applications. Sometimes, painting



FIG. 62.

*Erythema Serpens (Erysipeloid).*

the lid margin with 0.5% each of crystal violet and brilliant green in water is also helpful, together with small doses of X rays.

Chronic Paronychia. See p. 186

Bullous Impetigo of the new born. See p. 186

#### SECTION IV ERYTHEMA SERPENS (*Erysipeloid*)

In acute infection of the skin and deeper tissues by *Erysipelothrix rhusiopathiae* the organism which causes swine erysipelas. It was first described as *Erythema serpens* by Marrant Baker (1873. *St. Bart's Hosp. Reports* 9: 196) many of whose cases occurred in workers in Smithfield meat market. Later in 1887 Rosenbach described cases under the name *Erysipeloid* and investigated the bacteriology of the disease. It occurs usually in those who handle meat, poultry rabbits or fish as a result of infection of cuts and scratches on the hands and in fishermen from the bites of crabs and lobsters. The causal organism *Erysipelothrix rhusiopathiae* (*E. sui*) seems to be very widely distributed in nature.

The disease appears in 1 to 5 days after infection as a dull red or purplish area surrounding the scratch. This has a very slightly raised, clearly defined margin which spreads slowly outwards, eventually reaching a distance of 2 to 3 inches from the original site which by this time may have cleared up. The infected area is bluish red or purplish in colour slightly swollen it aches, burns or itches and there is often a peculiar feeling of tension, aggravated by heat. There are usually no constitutional symptoms but in cases reported by Klander in fishermen there were severe, deep-seated pain and throbbing lymphangitis and lymphadenitis with fever and malaise. In some of these cases an entire arm or leg was involved. Fatal cases are very rare but have been recorded. Accidental infections in laboratory workers may be severe.

The ordinary case is limited to a finger or two and part of one hand though infection may be transferred to the face or ear



FIG. 81

[Multiple *Lupus Vulgaris*, following manacle.

**TREATMENT** The disease is sometimes self-limited and tends to clear up in 3 or 4 weeks but penicillin or tetracycline (p. 33) cures it in a few days. There is an immune serum which may be used in severe cases (25 c.c. intramuscularly). As a local application 10% of ichthyol in glycerine seems to do well. Bedford (*Brit. Jour. Derm. & Syph.*, 1932, 44, 368) recommends 40% of ichthyol in petroleum jelly.

## SECTION V TUBERCULOSIS

Tuberculosis of the skin occurs in two principal forms, lupus vulgaris and scrofuloderma. Lupus vulgaris is a very slowly progressive, usually non-ulcerative tuberculosis of the skin characterised by *miliary tubercles forming lupus nodules* in the dermis. Scrofuloderma is a more rapid, and usually ulcerative, process taking place in the skin over broken-down tuberculous glands or joints. Here the tuberculous infiltration is diffusely distributed through the dermis instead of being localised in nodules, and a secondary staphylococcal infection takes a share in the process.

Lupus vulgaris usually occurs on the face and neck, although the buttocks are often, and any part of the body may be, affected. It is much commoner in hospital than in private practice and usually begins in childhood or adolescence. It is commoner in women than in men, and is much more frequent in the northern than in the southern countries of Europe, probably owing to the lack of sunlight in the former. There is often a family history of tuberculosis. The tubercle bacillus reaches the skin (1) by direct inoculation. Sequeira thought that picking the nose and picking impetigo lesions are common causes. The child may inoculate its buttocks with the bacillus by sitting on dried tuberculous sputum. Very rarely it follows BCG vaccination. (2) By extension of infection to the skin from underlying tuberculous glands or joints. (3) By blood borne tubercle bacilli from some broken-down gland giving rise to multiple lupus which occurs especially after meninges (Fig. 84)





over the lupus area may be either thinned or thickened and warty (lupus verrucosus). The latter type is usually found on the extremities.



FIG. 26

Lupus vulgaris. Nodules on both cheeks and tip of nose.

**CLINICAL FEATURES** Lupus vulgaris commonly begins on the nose or cheeks as a dusky red patch which causes no symptoms. On pressure with a watch glass the lesion becomes white and the "apple-jelly nodules" appear as small brown semi-transparent spots 1-2 millimetres in diameter. They have to be distinguished from freckles and from brown crusts on the surface. The mucous membrane of the mouth or nose is affected in a large proportion of the cases. The disease extends slowly but surely and in most cases has no tendency to spontaneous cure. Ulceration may occur and occasionally the whole area gets infected with impetigo. If the nose is attacked it is the alae and the cartilaginous tip which are destroyed. Eventually after many years, a large area of the face or neck is affected and is covered by a thin scar containing scattered lupus nodules while at the periphery of this there are more nodules and less scar tissue. Deformities of the nose, mouth and eyelids may

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be produced and carcinoma frequently occurs in old-standing areas of lupus vulgaris.

*Verruca neogenica* ("post mortem wart") is a small patch of warty lupus, usually on the hand, occurring as the result of inoculation of tubercle bacilli in an otherwise non-tuberculous person. The treatment is that of lupus and the prognosis is good.



FIG. 87

Lupus vulgaris. "Chin strap" type

**PROGNOSIS.** This is now good. Although certain long established cases do not yield entirely to the newer methods of treatment even they can be very materially benefited. The average fresh case clears in 6 to 9 months. The relapse rate has yet to be established but appears to be low. In multiple lupus, following measles or other acute illness, the majority of the patches usually heal spontaneously but leave one or two which become chronic.



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skin in a month than lupus vulgaris does in a year. The nodules of tertiary syphilis are larger than those of lupus vulgaris, are not translucent, and tend to be confined to the edges of the lesions the healed scar being free from them. The edges of tertiary syphilitic lesions tend to be serpiginous while those of lupus vulgaris are irregular. If the nose is attacked syphilis goes for the bones of the bridge, causing a saddle nose, while lupus vulgaris attacks the cartilages of the tip causing a beaky nose. Too much reliance should not be placed on the Wassermann reaction as both syphilis and lupus vulgaris may be present together and moreover 30% of tertiary syphilitics give a negative Wassermann reaction.

**TREATMENT GENERAL.** Although lupus vulgaris is tuberculous, the disease is rarely present in other organs and the patients are in good health. Apart therefore from normal attention to diet and hygiene no particular general measures are called for.

**MEDICINAL TREATMENT.** Isoniazid (isonicotinic acid hydrazide) is nowadays the standard treatment. It is given by mouth in doses of 3-6 mg. per kg. body weight, the average dose for an adult being 100 mg. 2-3 times a day. Improvement occurs within a month, and small lesions may disappear entirely in 6 months. Treatment should however be continued for two months after all clinical evidence of the disease has disappeared. In widespread cases, treatment may be continued for 18 months or longer if proving effective. Toxic effects are rare although increased or reduced appetite constipation, eosinophilia difficulty in starting micturition, headaches and dizziness have been reported. They usually disappear on continuing the drug, or reducing the dose, and always on stopping treatment. An occasional case of pellagra (nicotinic acid deficiency) has been described.

Streptomycin is the second most important drug. It is only necessary in those cases which fail to respond to isoniazid and then should be given in conjunction with that drug since alone it has little effect on lupus vulgaris. In most cases 1 gm. twice weekly proves sufficient but sometimes this dose has to be given daily. A higher dose should not be used.

Calciferol is now very rarely employed. Its introduction however by Dowling and Prosser Thomas in this country and Charby in France in 1945 and 1943 respectively was responsible for one of the most dramatic therapeutic revolutions in medicine. It is given by mouth in doses of 50 000 units three times daily for a month and thereafter twice daily for a year or more. Apparent cures are obtained in three quarters of the patients but relapses occur in two thirds of these. The toxic effects are gastrointestinal (indigestion, anorexia constipation, nausea and vomiting) neurological (headache photophobia and coma), renal (polyuria thirst and renal damage) malaise and loss of weight. They are associated with hypercalcaemia and all but one disappear on cessation of treatment. The single exception is renal damage, which may be prolonged or even permanent.

It appears that streptomycin and isoniazid inhibit or destroy the tubercle bacillus by direct interference with its metabolism, whereas calciferol has no direct effect on the bacillus but accumulates locally in the skin and stimulates the cellular reaction of the host to the chemical fractions of the tubercle bacillus, thus causing a fibrosis. Van der Lugt (1952) has shown that the action of the Flinsen lamp is similar to that of calciferol given by the mouth in that it increases the local concentration of Vitamin D and leads to the replacement of the tubercles by scar tissue (G B Dowling and G Wetherly Mein.)

LOCAL TREATMENT is rarely necessary nowadays. However chemical cautery of certain chronic and fibrotic lesions with acid nitrate of mercury painted over the lesion or pricked into the nodules is sometimes helpful. Similarly ultra violet light (p 42) can be an adjuvant to medicinal treatment in unresponsive cases.

**Scrofuloderma (Tuberculosis colligativa)** As stated above this is a more active form of tuberculosis of the skin consisting in a diffuse infiltration with tubercle bacilli and cells. The affected skin readily breaks down and the surface is usually infected with staphylococci. It begins as a bluish red swollen painless area usually situated over broken-down tuberculous glands. When the skin breaks down an ulcer is formed with irregular undermined bluish edges. The base of the ulcer is

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covered with pale granulations. Lupus vulgaris may follow or extend from the edges of a patch of scrofuloderma.

**TREATMENT** This is mainly that of the underlying tuberculous glands, or other tuberculous focus, combined with that described for lupus vulgaris. Scrofuloderma is usually more amenable to treatment than is lupus vulgaris.



FIG. 89

*Erythema induratum scrofulosorum.* (Dakin & Dacoe)

**Tuberculides** These are lesions of various types occurring in persons who have a tuberculous focus somewhere in the body usually in bones or glands. They are believed to be produced by the deposition of tubercle bacilli in the small blood vessels, where they are broken up and liberate their endo-toxins. This requires that patients who exhibit tuberculides should be allergic and should also have a considerable degree of resistance to tuberculosis, for otherwise presumably the bacilli would

multiply in size and multiple lupus would result. As it is, the lesions exhibit various degrees of tuberculous structure, epithelioid cells, giant cells, small round cells and plasma cells



FIG. 90

*Erythema induratum*. Whitfield type.

being present, but tubercle bacilli are not, or are only very rarely found. The commonest forms of tuberculides are erythema induratum scrofulosorum (Bazin's disease) papulo-necrotic tuberculides and lichen scrofulosorum.



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*Erythema induratum scrofulosorum* (Bazin's disease) This consists of subcutaneous nodules, which subsequently reach the surface, turn purple and may break down, situated on the lower



FIG. 91

Bazin's Disease. Ulcerated type. Note resemblance to tertiary syphilis, but Wassermann reaction was negative and anti-syphilitic treatment without effect.

half of the leg in young women. They begin as cellular infiltrations around the venules in the subcutaneous fat and gradually extend into the skin and so to the surface. They are somewhat tender and the early nodules can be felt before they are seen. When they have become visible they are purple in colour and are usually found on the back but also on the sides and front of the lower half of the leg. Girls who exhibit these nodules are usually rather stout with thick ankles, coarse skins and prominent, red, hair follicles on the legs. They often suffer from some degree of acrocyanosis, i.e. cold bluish red hands and feet (p. 76). The disease is generally worst in cold weather and often appears in several succeeding winters getting well in the

summer. There is usually a history or some evidence of tuberculous glands. Many of the lesions clear up without leaving scars, but if they break down they form indolent punched-out ulcers resembling gummata. Such ulcers leave depressed pigmented scars not unlike those left by gummata.

**DIAGNOSIS.** Erythema induratum is a syndrome with many causes. Tuberculosis is only one of them and nowadays the least common. In the majority of cases the condition is an example of perniois (p 74), though here the ulceration is less common, severe or persistent. The condition also occurs in older women without any evidence of perniois. In this group the nodulation is more painful, inflammatory and evanescent and ulceration seldom occurs. The cause is unknown, but for clinical and histological reasons it is frequently referred to as nodular vasculitis. Other



FIG. 92.

L. A. J. J. J. J. J.

Papulo-Xanthic tubercules. Not white scars as well as active lesions.

cases are seen from time to time which do not fall into any of these groups.

From *tertiary syphilis* (p 261) This generally occurs in older people is usually asymmetrical and is not likely to be confined to the lower half of the leg. Other signs of syphilis may be found. The Wassermann reaction will probably be positive, and the effect of anti-syphilitic treatment will usually be rapid.

From *varicose ulcer* (p 276) This again usually occurs in people and is generally situated on the inner side of the older lower third of the leg. It is usually single and the varicose veins will probably be obvious. There is likely to be a history or evidence of chronic varicose dermatitis preceding the ulcer.

From *Erythema nodosum* (p 203). This is an acute febrile

disease with painful tender swellings appearing suddenly on the front of the upper part of the leg. The swellings are redder than those of erythema induratum and never break down.

**TREATMENT** of Bazin's disease. This is the same as described for lupus vulgaris. The perniotic element, however must also be dealt with (see p. 75). The ulcers themselves may be dressed with Eusol or because of the frequent secondary infection, a local antibiotic.

**Papulo necrotic tuberculides** are small papular lesions which, as their name implies, undergo necrosis in the centre they eventually disappear leaving very characteristic oval depressed scars, 2-3 millimetres in length. They are rather like small boils but are more purple in colour are painless and very indolent, taking from two to eight weeks to run their course. They tend to appear symmetrically in crops, and the areas most affected are the extensor aspects of the hands arms elbows, shoulders, buttocks, legs and feet. The face also is often affected. They most commonly occur in young adults between 20 and 30 years of age, who often have acrocyanoma. Like Bazin's disease they tend to be worse in winter.

**PROGNOSIS.** They always get well but are liable to recur so long as the tuberculous focus persists.

**DIAGNOSIS.** From boils (p. 187) they are distinguished by occurring symmetrically in crops, and by their painlessness, indolence and characteristic scars.

From broken chilblains (p. 75) by their regular size and shape (which also applies to the scars they leave) their long duration, and by the absence of irritation. Chilblains are irregular in size, shape and distribution itch violently and if broken are painful and leave irregular scars. Their duration also is much shorter.

**TREATMENT** As for lupus vulgaris.

**Lichen scrofulosorum** An eruption consisting of patches of small rounded or acuminate papules mostly about the hair follicles, giving the appearance of permanent goose skin. It occurs in tuberculous children or adolescents, usually on the front and sides of the trunk, but sometimes on the extremities.

The papules may be of normal skin colour or else reddish or brownish. The summit of each may bear a small scale or spine, or may contain a bead of pus. The eruption may appear suddenly and persist for months or years. It may then disappear without trace or may leave minute scars. The eruption causes no symptoms and is often unnoticed by the patient.

**PROGNOSIS.** Lichen scrofulosorum always disappears in time but may recur so long as the primary tuberculous focus persists.

**DIAGNOSIS.** From *lichen planus* (p. 361). In this the papules are flat topped, polygonal and shiny usually violet-coloured and may itch considerably. They may often be found in a typical form on the front of the forearms or wrists, and white spots and streaks may sometimes be seen inside the cheeks. From the *lichenoid trichophytide* (p. 179). This may be very like lichen scrofulosorum but a deep-seated inflammatory ringworm is present as the original focus. From *follicular eczema* (p. 322). This most commonly occurs on the limbs, usually itches a good deal, and some of the lesions will probably be vesicular or crusted. From the *lichenoid syphilide* (p. 254). This most commonly occurs in adults, and other signs of syphilis including a positive Wassermann reaction will probably be present.

**TREATMENT.** As for lupus vulgaris.

**Rosaceous Tuberculide.** In this rather uncommon condition the lesions are minute papules or very small, sometimes follicular pustules. They are situated on the sides of the cheeks, forehead, chin and neck. The centre of the face is rarely affected, in distinction from true rosacea, and there is no excess of sebum. The papules are red to brownish yellow in colour and glass pressure reveals a small brown or yellow nodule at the site of the papule. The intracutaneous tuberculin test is usually strongly positive and the response to the injection of gold salts is often good.

## SECTION VI ANTHRAX

A relatively rare disease which is important on account of its high mortality. It is caused by infection with the spore-forming

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**B Anthrax** and occurs in three forms cutaneous (malignant pustule) pulmonary (wool sorters disease) and rarely intestinal.

**ETIOLOGY** Primarily a disease of animals, it may be conveyed to man by direct contact or via hides, wool, hair or shaving brushes made from the latter. In the cutaneous form the bacillus enters through a cut or scratch.

**SIGNS AND SYMPTOMS OF CUTANEOUS ANTHRAX** The site is usually the face the back of the neck (from carrying hides) or the forearms. A few hours after inoculation there is itching at the site. In one to three days a papule forms which rapidly becomes a vesicle containing blood stained fluid and surrounded by an area of inflammation. Central necrosis then occurs, the typical "malignant pustule" being present one and a half to three days from infection. This is a central black eschar surrounded by a ring of vesicles outside which is an area of intense inflammation with subcutaneous oedema. The appropriate lymphatic glands are enlarged. Pain is usually slight. General symptoms are slight at first, but if the lesion is not recognised and treated they soon become severe with high temperature (104°-105°) and collapse.

**DIAGNOSIS** The patient's occupation with hides, wool, hair or animals should lead one to consider the possibility as should also the recent purchase of a cheap shaving brush. The diagnosis is confirmed by the finding of anthrax bacilli in the lesion, which, however is not always easy.

**PROGNOSIS** In the absence of treatment death may occur in 3-5 days but mild cases which recover spontaneously are sometimes seen. The mortality is from 5% 25% being highest when the lesion is on the face.

**TREATMENT** That recommended by A. B. Christie (*Practitioner* 1953 171 673) is by penicillin, one million units on admission followed by quarter of a million units six hourly for seven days. Or aureomycin may be given by mouth 500 mgm. six hourly for three days followed by 250 mgm. six hourly for four days. Christie states that arsenic and sulphonamides have now no place in treatment. The pustule should not be excised.

If the patient is severely ill, he may have glucose saline or blood intravenously and if apprehensive, morphine,  $\frac{1}{4}$ – $\frac{1}{2}$  gr at the start.

Hutch considers it highly important to keep the affected part at rest by splint or sandbags.

with a yellow crust. Fresh lesions are constantly appearing on different parts of the face and the outline of each lesion is a smooth curve. In herpes the vesicles are small, tense, uniform and do not increase in size once they have appeared. They



FIG. 93.  
Herpes simplex.

tend to dry up rapidly into small pinhead-sized yellow crusts on a red base. If they become confluent forming a yellow patch the outline of this is polycyclic. There is generally only one patch or a small group of patches close together. There is very likely a history of previous attacks. Impetigo, however, is frequently implanted on a patch of herpes. From *herpes zoster* (p. 200). When this occurs on the face it is generally in the distribution of one of the branches of the fifth cranial nerve usually the supra-orbital. In any case it is unilateral whereas herpes simplex is often bilateral. Herpes zoster is generally preceded for a day or two by pain and is accompanied by swelling of the lymphatic glands in the neighbourhood. Previous attacks are very rare. In herpes zoster especially

supra-orbital, the vesicles are often haemorrhagic and considerable scarring is left.

Herpes genitalis from *chancroid* (soft sore) In this the ulcer has an inflamed red margin, overhanging edges and a cribriform base covered with a yellow slough. One or more of the inguinal glands are usually inflamed, tender, adherent to skin and deep tissues, and may suppurate. Herpetic lesions, however may become infected by *Ducrey's bacillus* and converted into *chancroids*.

From *primary syphilis* (p 345) Herpes genitalis bears little resemblance to a primary sore except when the ulcers are healing, when there is occasionally a good deal of induration immediately beneath and around them. Typically there is no induration. In suspicious cases repeated examinations for *Sp pallida* must be made, and if they are not found several Wassermann reactions must be performed—at first at weekly then at fortnightly and finally at monthly intervals. It must be remembered that the Wassermann reaction does not become positive for from two to five weeks after the appearance of even a genuine primary sore. The incubation period may be of some assistance if all the dates of coitus for the previous month or five weeks are considered. Herpes usually appears one to two days after coitus and a primary sore from two to five weeks after. In a case of herpes genitalis there will very likely be a history of previous attacks. Again it must be remembered that herpetic sores are easily inoculated with *Sp pallida* and that a penile lesion which is obviously herpes when seen, may later develop the characters of a primary sore.

TREATMENT of herpes simplex. There is no specific treatment and all that can be done is to keep the ulcers clean and dry. Mopping frequently with spirit of camphor "T C P" and Alibour (p 37) or "Milton" (solution of sodium hypochlorite) and powdering with zinc oxide calamine or talc powder is usually the best method. Unfortunately there is no known treatment which will certainly prevent the recurrence of herpes. Inoculation of serum from a recent vesicle on to a lightly scarified site on the arm appears to have prevented further attacks for some years in a few of my cases. Weekly vaccination on the arm for



about 11 times with ordinary vaccine lymph is said to have cured recurrent herpes on the face. Occasionally the removal of a troublesome tooth or other source of irritation will be followed by an interval of relative freedom. If discovered before the



FIG. 94

Kaposi's varicelliform eruption, due to herpes simplex virus in an infant suffering from infantile eczema. Note umbilicated pustules. Child recovered.

vesicles have begun to appear an attack can occasionally be aborted by a small dose of X rays or by painting with silver nitrate, but the latter causes a disfiguring stain, and in any case the outbreak is usually well under way by the time the patient



FIG. 93.

Herpes zoster

wakes in the morning, so that it is too late to consider abortive treatment. A small dose of X rays, however, often shortens the course of an existing herpes, and lengthens the interval before the next attack.

#### Kaposi's Varicelliform Eruption

Sometimes the primary infection of herpes simplex manifests

itself as a more or less generalized cutaneous eruption consisting of the characteristic umbilicated vesico-pustules. This condition is seen almost exclusively in individuals, usually infants and young children, suffering from atopic eczema, the herpetic lesions occurring on the eczematous areas. In severe cases there is high fever and grave systemic symptoms and the malady may be fatal. A similar condition can be caused by the vaccinia virus in atopic individuals and is the reason why vaccination should be postponed until the eczema has cleared up.

Treatment consists of general supportive measures, systemic antibiotics to control the massive secondary infection of the lesions with staphylococci and injections of specific herpetic or vaccinal immune globulin.

### HERPES ZOSTER. ZOSTER, ZONA SHINGLES

An outbreak of vesicles, preceded by pain and accompanied by enlargement of neighbouring lymph glands in the distribution of a sensory nerve on one side of the body associated with inflammatory changes in the corresponding posterior root ganglion



*Sir E. Graham Little, Case*

FIG. 96

Herpes zoster. Left III and IV cervical nerves.

**ETIOLOGY** Zoster is commonest in adults but may occur at any age. It sometimes seems to occur in small epidemics but direct case to case infection is very uncommon. In about 4% of cases, it occurs in association with a reticulosis. In a large number of recorded cases an attack of zoster in an adult has been followed by varicella 14-16 days later in child contacts. The converse sequence, varicella—zoster is ten times less common. Zoster and varicella sometimes occur concurrently in the same patient, but on the other hand zoster may follow a few weeks after an attack of varicella, when one would expect the immunity to be high. Inoculation of the contents of zoster vesicles into children under five years of age has in a number of cases (Kundratitz, Bruusgaard) given rise to varicella. The sera of zoster and varicella patients contain identical antibodies (Netter and Urbain, Bram). It seems quite clear that the two diseases are caused either by the same virus or by two closely allied viruses which often occur together. The appearance of the vesicles indicates the arrival of the virus in the skin after its journey down the sensory nerves. Zoster is always the same disease, whether it is "spontaneous" or follows injury or the taking of arsenic or other drugs, and any case may give rise to varicella.

**HISTOPATHOLOGY** The vesicles are situated in the epidermis — and are characterised by the presence in them or in their walls and floors, of peculiar swollen, degenerated prickle-cells called "balloon cells." According to the classical work of Head and Campbell the nerve changes in zoster consist in

1. In the posterior root ganglion acute inflammation with haemorrhages and round cell infiltration, leading to destruction of the ganglion cells and eventual sclerosis, corresponding exactly with the changes observed in the anterior horns in acute anterior poliomyelitis.

2. Degenerative changes spreading upwards into the posterior columns of the cord and downwards along the peripheral afferent nerves.

3. Degeneration in the nerves of the deeper layers of the corium beneath the vesicles, consisting in swelling of the

neurilemma, degeneration of the myelin sheath and monileform swelling of the axis cylinders.

In comparatively rare cases the inflammatory changes spread horizontally in the cord from the posterior to the anterior horns and an associated motor paralysis then occurs.

**CLINICAL FEATURES** The usual course of a case of zoster is that the patient complains of pain for a few days in the distribution of a sensory nerve on one side usually one of the spinal nerves in the thoracic region. After two or three days of this there may be some malaise and slight fever. Then a red area



Dr. J. S. S. S. S. S.

FIG. 97

Herpes zoster

appears and the pain eases off somewhat as the vesicles appear on the third or fourth day. Lymphatic glands in the axilla or neck are usually enlarged and often tender. The vesicles are at first small (2 millimetres) tense, and filled with clear fluid, they tend to run together and after a few days become purulent. Sometimes especially on the forehead, they are haemorrhagic. After a week or so they dry up into scabs and in another week fall off leaving small scars. On the forehead and scalp in supra-orbital zoster there may be much necrosis and severe scarring. In young patients there are usually no sequelae but in elderly persons zoster may be followed by severe and intractable neuralgia in the affected nerve.

In supra-orbital zoster if there are vesicles on the side of the nose it indicates that the ciliary ganglion is involved and a



FIG 94.

Herpes zoster affecting the first division of the fifth cranial nerve (Herpes Ophthalmicus). Note lesions on side of nose (involvement of ciliary ganglion).

watch must be kept on the eye for conjunctivitis and corneal ulceration. Sometimes "aberrant vesicles" are seen scattered about the trunk or limbs. These may be numerous and indistinguishable from the lesions of varicella. An attack of zoster usually confers lifelong immunity

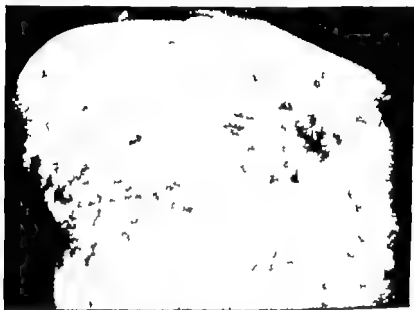


FIG. 99

Pigmentation and scarring left by Herpes zoster. This patient, set. 62, had severe post-herpetic neuralgia, which was relieved by radiant heat.

**PROGNOSIS** The disease usually lasts about two weeks and recovery is complete. In elderly people the possibility of intractable neuralgia following zoster should be remembered. In cases of supra-orbital zoster there is the possibility of permanent damage to the eye and the probability of permanent scarring on the forehead

**DIAGNOSIS.** In the ordinary case this is easy. The diagnosis from herpes simplex on the face has been considered on p. 221. Difficulties are most likely to arise in cases which are seen before the vesicles have appeared when there is only pain or a painful

red patch. If the possibility of zoster is borne in mind the position of the patch or pain in the distribution of a cutaneous nerve on one side of the body only will probably lead to a correct diagnosis.

**TREATMENT** There is no specific treatment. Protection of the vesicles from trauma and infection, and relief of pain, are the requirements. If seen in the stage before the appearance of the vesicles, or while the latter are still small, the area may be painted with collodion strengthened with thin layers of cotton wool. If the vesicles are already large or have ruptured, local antibiotics are the best application, since they control secondary infection and so reduce residual scarring. The pain may require aspirin, phenacetin, etc., by the mouth or even injections of morphia while radiant heat and ultra-violet light locally are often helpful. Intramuscular injections of vitamin B<sub>12</sub> (cyanocobalamin) in daily doses of 1000 micrograms for 7-10 days, are said to shorten the course of the disease and decrease the chances of subsequent neuralgia. One c.c. of pituitrin (posterior lobe) intramuscularly on alternate days sometimes relieves the pain. If the lesions become infected they must be treated by local antibiotics while ultra violet light will help in overcoming the sepsis. For the treatment of neuralgia following zoster X-rays are useful in some cases either superficial to the affected skin or deep to the appropriate ganglia. Irradiant heat is also helpful.

### WARTS (VERRUCAE)

Localized hypertrophy of the prickle cell layer of the skin due to infection with a filterable virus leading to secondary enlargement of the papilla. The following varieties of warts are described: 1 Plane 2 Common 3 Plantar 4 Filicoid

Digitate 6 Condylomata acuminata

The essential histology of all these types of warts is a hypertrophy of the prickle cell layer (anthosis) due to the fact that this layer undergoes hyperplasia under the stimulus of a filterable virus. (Normally the prickle cells in the epidermis are in the basal layer and the prickle cells once formed



increase in number) The hypertrophy of the prickle-cell layer leads to an increase in the length and thickness of the inter papillary parts of the epidermis which pull up the distal parts of the papillae and also push themselves downwards between their bases. The net result is a lengthening of the papillae. In certain types of wart (plane, common, plantar and filiform) there is also an increase in the thickness of the horny layer. In



FIG. 100.

Plane Warts on hand.

filiform and digitate warts and especially in condylomata acuminata, the hypertrophy of the prickle-cell layer causes the papillae to be stretched out enormously and to project from the surface. In these types there is not much thickening of the horny layer. In fact in condylomata acuminata the latter is particularly thin.

**ETIOLOGY** Numerous observers have shown that a filtrate of ground up warts, if inoculated intradermally will cause the appearance of warts at the site of inoculation after an incubation period of some weeks or months. It also appears that all the varieties of wart mentioned are due to the same virus, for



FIG 10L

Plaque Warts on face Note occurrence along lines of scratches.

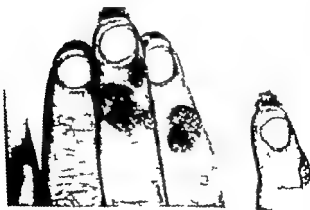


FIG 11J

Common Warts.

## 234 DISEASES DUE TO FILTERABLE VIRUSES

inoculation of a filtrate of common warts may produce plane warts or common warts, and a filtrate of condylomata acuminata



FIG. 103.

Planter Warts.

may produce common warts. There is also plenty of clinical evidence to show that warts are contagious and auto-inoculable

No doubt a predisposition on the part of the patient, depending on some factors which are not understood, is also necessary witness the way in which warts often disappear spontaneously or can be "charmed away" For an account of popular charms for warts, see Rolleston, J. D., *Brit. Jour. Derm. and Syph.*, 1940 52, 43

**Plane or juvenile warts.** Plane or juvenile warts which occur in great numbers on the hands and faces of children, and sometimes of adults, are the simplest type of wart. They are small, smooth elevations 2-3 millimetres across and perhaps half a



FIG 104  
Plaster Warts. Multiple small type.

millimetre high, and are the colour of the normal skin or sometimes slightly brownish. When examined with a lens the surface can be seen to be not truly uniform but to show a slight degree of "raspberry" appearance.

The common wart is a further development from the plane wart in the direction of increased length and irregularity of the papillae, and increase of horny layer. Common warts usually occur on the hands where they may reach a diameter of half

an inch and a height of a quarter of an inch. They are often blackened by ingrained dirt. The tops of the individual papillae, each with its cap of horny layer, can be made out by the naked eye.

The plantar wart is similar to the common wart but as it occurs on the ball of the foot or on the heel, it is being constantly pressed into the skin by the weight of the body and so does not project much above the surface. It does project deeply into the corium however and, pressing on the nerve endings there is acutely tender. Plantar warts are common in schools, where the infection is probably picked up by the bare feet off bathroom floors. In patients with a flattened transverse arch warts often occur in the centre of the ball of the foot. They are often mistaken for corns. A corn (p. 72) consists purely of a thickening of the horny layer and contains no blood vessels. Its surface is smooth and hard. A wart contains blood vessels which can be seen and may bleed if the surface is carefully pared, and the tops of the constituent papillae can generally be made out on careful examination.

The filiform wart is a single structure like a stout thread projecting from the skin. It may be an eighth to a quarter of an inch in length and is usually of normal skin colour with a horny tip. These warts are usually seen on the neck in middle-aged women but may occur on the nostrils or on the eyelids.

Digitate warts are most often found on the scalp, but small ones are sometimes seen on the beard area in men. They are like a cluster of filiform warts diverging from a narrow base. They are often caught by the comb or razor and bleed easily.

Condylomata acuminata occur on the genital organs or about the anus in both sexes. They are sometimes called venereal or gonorrhoeal warts. The term venereal is probably justifiable but "gonorrhoeal" is not, as they have no relation to gonorrhoea, except that they may be contracted on the same occasion. Gonorrhoea then appears in 3-4 days, and the warts after some weeks or months, as their incubation period is so much longer. They are like digitate warts, but may be much longer and often occur in enormous numbers, especially

on the vulva. In colour they are pink, as their horny layer is thin and they are very vascular

**TREATMENT OF WARTS PLANE WARTS.** A large number of internal treatments have been recommended, e.g. lime water half a pint (235 c.c.) daily for three weeks, Mag sulph. in sufficient quan



FIG. 103.

Multiple Warts. Digitate and Filiform Types.

tity to cause three liquid stools a day for three weeks, an eighth to a quarter of a grain (0.008-0.016 gm.) of green iodide of mercury thrice daily for two months, and the injection of N.A.B. or sub-farsenol. One has to remember that warts often disappear spontaneously and quite quickly for no ascertainable reason. Local treatment is required sometimes. The mildest is to apply a salicylic acid and hydrarg. perchlor. lotion (p. 389) or a salicylic acid ointment (2-4%). The quickest is to freeze the warts with the ethyl chloride spray and then scrape them off, mopping the raw surface with a 3%-4% silver nitrate solution. Instead of scraping the warts may be destroyed with the galvano-cautery electrolysis, zinc ions or diathermy though some scarring may occur after the latter. X rays—300-400r sometimes remove them.

**COMMON WARTS.** The quickest way again is to freeze with ethyl chloride and scrape away mopping the raw surface with silver nitrate solution as above. If the wart is very large novocaine may be used instead of ethyl chloride. Caustics such as nitric acid and liquid acid nitrate of mercury are also effective. They should be applied carefully to the wart only at intervals of three days. The layer of tissue killed by the previous application must be cut away before another is made. Freezing with CO<sub>2</sub> snow or better still, with liquid oxygen or nitrogen (p. 38) is more satisfactory than chemical cautery. Zinc ions are a clean and effective way of curing warts if the apparatus is available. X rays radium and radon are useful for some obstinate warts, especially about the nails.

**PLANTAR WARTS.** About 5 per cent disappear spontaneously. Of the manifold possible treatments, none are effective in more than 80 per cent of cases or in less than 50 per cent. Probably the most effective is to use a general or local anaesthetic according to the number of warts and type of patient and to scrape the wart right out with a Volkmann's spoon. This leaves a smooth white cavity of surprisingly large size. The overhanging edges of this are then clipped away and the cavity plugged with gauze soaked in 2%-4% silver nitrate solution or tinct. iodi. A dry dressing is applied over this. In a couple of days the dressing is renewed and

less or no plugging inserted. By the end of a week or two the hole will have filled up. There is surprisingly little pain after this operation, and many patients can walk about quite well even the same day although it is advisable to rest the foot for a day or two. If properly done it seldom fails to cure the wart, and it never leaves a painful scar.

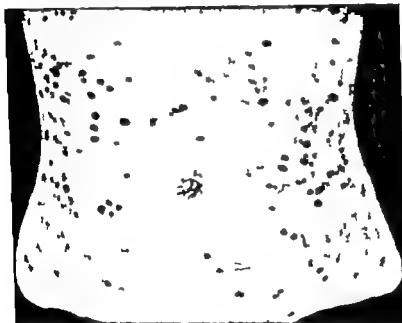


FIG. 106.  
Seborrheic Warts.

Forty per cent salicylic acid plaster or 25% podophyllin in liquid paraffin applied nightly to the wart under elastoplast for 3-6 weeks are other good methods. Bathing the wart in 5% formalin in a saucer protecting the surrounding skin with vaseline for 5-10 minutes nightly is less effective. In each of these three methods the wart should be pared weekly with a scalpel. Freezing has to be prolonged to be successful and usually causes undue pain. X rays can be helpful in carefully



selected cases, but should never be given other than by a highly skilled and experienced radiotherapist and should never be given more than once

**FILIFORM WARTS** should be twisted off or cut off with a galvano-cautery. Or the base may be transfixed with an electrolysis needle to thrombose the vessels and the wart then cut off or allowed to fall off.

**DIGITATE WARTS** are best treated with glacial acetic acid. They absorb this acid readily and one application is usually enough. J. V. Macgregor (*B.M.J.* 1945 1 593) reports good results in warts on the beard region with podophyllum (see below)



FIG. 10  
*Molluscum contagiosum*

**CONDYLOMATA ACUMINATA** A very useful treatment introduced by Culp Magid and Kaplan (*J Urol* 1944 51 655) is as follows. A 25% suspension of podophyllum resin in liquid paraffin or a paste of podophyllum and water is applied thoroughly to the warts or to the whole warty area for 8 hours at a time. It is then washed off and a dusting powder applied. The warts shrivel up and turn yellowish in 24 hours and fall off in two or

three days. In cases with many or large warts repeated applications may be required but the suspension should not be left on for more than 8 hours at one time as it may set up a dermatitis. Older methods of treatment are cutting off the warts with a galvano- or diathermy-cautery under a local or general anæsthetic. They may be cut or scraped off and the bleeding area mopped with silver nitrate solution, but they bleed very freely and the cautery is preferable. If small, merely rubbing with the silver nitrate stick is often enough. This is the only type of

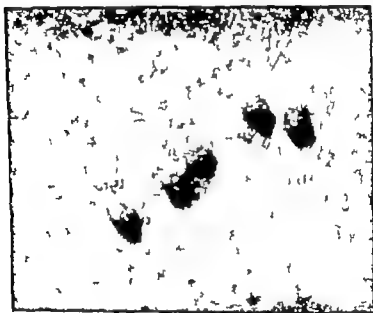


FIG. 108.

*Molluscum contagiosum.*

wart for which the silver nitrate stick is of any use, in my experience

"Seborrhoeic" or senile warts. These result from a localized hypertrophy of the basal cell layer of the epidermis. They are not caused by a virus, but are included here for convenience. They

are flat, brown or black excrescences  $\frac{1}{4}$ - $\frac{1}{2}$  an inch in diameter raised only slightly above the general surface which are common on the trunk of patients aged forty five or over though they may occur as early as thirty. They may also occur on the temples or elsewhere.

The surface is horny and if pulled off leaves hypertrophied papillae projecting. They have to be distinguished from pigmented moles. Seborrhoeic warts are usually of shorter duration, and are present in greater numbers than pigmented moles. They give the appearance of being on rather than in the skin, and the way the surface can sometimes be pulled off is also a help in diagnosis. On the temples they must be distinguished from solar keratoses (p. 80) which may become malignant. Seborrhoeic warts never do this. Usually they cause no symptoms but occasionally they give rise to severe itching.

**TREATMENT** Painting with pure carbolic acid usually causes them to fall off in a week or ten days. If this fails CO<sub>2</sub> snow applied for a minute or so is effective.

Other warty types of lesion which may be mentioned here are solar keratoses (p. 80) the similar lesions due to long exposure to arsenic (p. 119) or tar (p. 103) and the post mortem "wart" (p. 208). None of these have any relation to the infective warts described in this section.

### MOLLUSCUM CONTAGIOSUM

Small pearly white epithelial tumours, due to infection with a filterable virus, in which the constituent cells undergo degeneration and form "molluscum bodies" with peculiar staining reactions. They may occur anywhere on the body and are often found on the trunk in persons who take Turkish baths frequently the infection being no doubt acquired from the bath-gown, slab or masseur. They are nearly always multiple and may be very numerous. *In size they vary from 2.3 millimetres to 2 centimetres.* The larger lesions are sometimes pedunculated. Their colour is pearly white or pink, and the surface is shiny except for a dull spot in the centre.

where the degenerated cells reach the surface. The incubation period is six to twelve weeks.

Histologically the tumour consists of a group of enormously hypertrophied interpapillary processes of the prickle-cell layer the cells in the centre of each process degenerating and staining different colours, as they get nearer the surface. This mass of hypertrophied interpapillary processes raises up the surrounding corium and epidermis to form the outer layers of the tumour.

**TREATMENT** The lesions may be removed by curetting, by the galvano-cautery by boring into each with a pointed match stick dipped in pure carbolic acid, or by slitting the tumours across and squeezing out the white, cheesy contents, subsequently applying tinct. iodi. The boring process with carbolic acid is the method usually preferred by patients. ■ W Barefoot was successful with podophyllum resin locally in the form of 20% solution in 95% alcohol painted on each lesion on alternate days for three or four weeks.

## CHAPTER XI

### SYPHILIS

SYPHILIS is a disease caused by the spirochaete *Treponema pallidum*, involving all parts of the body including the skin. The infection is commonly acquired in adult life but the syphilitic mother may infect the foetus in utero in which case the condition is spoken of as pre-natal or congenital.

The study of syphilis is such an enormous subject that only the principal features of its pathology and cutaneous lesions will be dealt with here. Information on its osseous, visceral, cardiovascular and nervous lesions, and on its treatment both in the acquired and congenital types, must be sought in works on Venereal Disease.

**Infection.** Close contact with an infected person is necessary for transmission to occur and this contact must be at a time when there are spirochaetes present on the patient's skin or mucous membranes. The necessary conditions are most likely to be fulfilled during sexual intercourse but being kissed by a person with lesions in the mouth or even the use of contaminated articles such as cups may cause infection. But the spirochaete does not survive drying and this fact prevents the common occurrence of extra-genital lesions.

The organism may enter the body through some small abrasion on the skin surface or even possibly through the intact skin, and spreads via the lymphatics to the regional lymph glands and via the blood stream to all the tissues of the body at an early stage. Thus the disease is not localised even in the primary stage.

**Pathology.** The essential pathology of all syphilitic lesions is the same. The presence of the spirochaete in the perivascular lymphatics produces an infiltration of plasma cells, lymphocytes, and mononuclear cells. This causes proliferation of the intima

of the small vessels, progressing in some cases to an obliterative endarteritis, with resulting tissue destruction followed by fibrosis. Varying degrees of inflammation and necrosis are seen at different stages of the disease, depending on the development of the patient's resistance and sensitivity (allergy) to the spirochaete.

The cellular infiltration gives rise to the firm induration characteristic of many syphilitic lesions, which are typically chronic, painless and indolent. The longer the disease has been present in the body the fewer are the organisms present in a lesion and the less likely the chances of contact infection. The primary chancre contains many spirochaetes, and they occur in very large numbers in the mucocutaneous lesions of the secondary stage, but not in the gummatous lesions of late syphilis.

The pathological changes in the skin take place primarily in the dermis, the epidermis undergoing changes consequent upon interference with its nutrition.

The spirochaete, *Treponema Pallidum*, is a slender spiral organism from 7 to 12 microns in length, showing about seven turns in its spiral. Staining methods are difficult, but it can be identified easily by dark ground illumination of a specimen of serum obtained from a lesion by exocration.

## EARLY ACQUIRED SYPHILIS

### Primary Stage

The incubation period between infection and the appearance of a sore may range from nine to ninety days, with an average of three to four weeks. In most cases there is a single lesion, but sometimes multiple chancres are seen, appearing simultaneously or within a few days of each other. The chancre begins as a red papule which enlarges to about the size of a pea and rapidly becomes eroded, giving rise to an ulcer with a clean, grayish or raw beef coloured base and a regular clearly defined border. On palpation the edge can be felt to be hard and indurated sometimes cartilaginous in consistency but induration is not



FIG. 100.

Early Syphilis. Primary stage. Chancre on Penis.

invariable. The serous exudate produced from the ulcer is extremely infectious, containing numerous spirochaetes.

The primary chancre is indolent, taking a slow course and healing spontaneously if untreated, in three to ten weeks, leaving a thin atrophic scar. The scar is not usually very noticeable, and may well be missed. The lesion is practically painless unless secondary pyogenic infection has taken place, in which case the sore and the regional lymph glands become tender. Considerable oedema of the tissues in the proximity of a primary chancre is not uncommon. Within a week of the appearance of the chancre the regional lymph glands become enlarged in the majority of cases. They are discrete, painless and of a rubbery consistency. With genital chancres the enlargement is often bilateral, but with extra-genital chancres the adenopathy may be unilateral.

The primary stage may be missed entirely in cases in which the infection results from deep inoculation of the spirochaetes, as from puncture wounds of a vein or blood transfusion with infected blood. This is known as "syphilis d'emblée". The first signs and symptoms are those of the secondary stage.

Ninety five per cent of chancres occur on the genitalia, the other five per cent on other parts of the body. These extra-genital chancres are found on the lip, the tongue, the tonsil, the finger, the eyelid, the nipple (a common site in the old days when wet nurses were employed), the anus and rectum or on any part of the skin surface. In each site they give rise to particular difficulties in diagnosis, which will be discussed later.

Genital chancre in the male may occur on different parts of the penis. The commonest site is the coronal sulcus, but the glans penis, the frenum, the urinary meatus, the shaft of the penis or the prepuce may be involved. In the latter case "button" induration may result, so that on retraction the prepuce is found to flap back suddenly as if a button were incorporated in it. A chancre may occur inside the urethra (intra-meatal chancre), in which case the only symptom may be urethral discharge. The clinical diagnosis may be made by palpation or by urethroscopy. A chancre occurring under the prepuce may give rise to a balanoposthitis with oedema so that the prepuce



will not retract. In some cases in which the shaft of the penis is involved the distal part of the penis may have been protected by a condom during sexual intercourse.

Genital chancres in women may occur on the labium majus or minus, at the fourchette, on the clitoris or near the urethral orifice they may also be found on the vaginal wall (rare) or on the cervix uteri. Chancres on the vulva are commonly accompanied by considerable oedema of the labia, which may be unilateral. During pregnancy the lesions may be larger and more indurated on account of the increased vascularity of the parts.

**DIAGNOSIS** This is made by a combination of clinical and laboratory examinations. In primary syphilis the most certain method is by identifying the *Treponema pallidum* in the serous exudate from the ulcer by dark ground examination. If initially negative dark ground examinations should be repeated daily for at least three consecutive days, while it may be necessary to search for a longer period, applying saline solution daily to the lesion. In cases in which an antiseptic or penicillin cream has been applied or in which the chancre is healing or is not visible (i.e. intrameatal or subpreputial phimotic chancre) gland puncture is particularly useful. By this technique 0.1 m.l. of saline is injected into an enlarged regional lymph node and some serum is then withdrawn and examined by the dark ground method. Serological diagnosis utilising the Wassermann and Kahn (or P P R) tests, is of limited use in the primary stage as fifty per cent of cases do not develop positive reactions till the third week and even after the fifth week ten per cent of cases may give negative results. However serological reactions should always be routinely determined and a quantitative record of the Kahn (or P P R) test is useful for following the progress of the disease and the efficacy of treatment.

**DIFFERENTIAL DIAGNOSIS** (a) Genital chancres Lesions occurring on the genitalia which may be mistaken for primary syphilis include chancroid (soft sore), the ruptured vesicles of herpes genitalis, erosive balanitis, scabies with secondary infection, gummatus ulceration and epithelioma. Lymphogranuloma, Herpes Zoster (sacral 3) granuloma venereum,

traumatic ulcers and secondary syphilis may also occasionally have to be considered in the differential diagnosis. In this country the incidence of chancroid is low. The lesions are usually multiple with an irregular edge, they are painful, bleed easily and are not indurated but there may be considerable tissue destruction. The inguinal glands, if involved, are enlarged, tender and matted together with redness of the overlying skin. This condition is termed a bubo and if untreated the suppurative adenitis will break down and discharge pus. Dark ground examinations and serological tests for syphilis are negative, and the intradermal Ito test (using Dmelcos vaccine) is positive if the lesions have been present for at least ten days. The absence of induration helps to distinguish an herpetic lesion, and on close examination there will be seen a collection of tiny ulcers representing the original group of vesicles.

A patient with scabies will give a history of itching, and other burrows may be found on the body in the typical distribution. A scraping from the genital lesions may demonstrate the *Sarcoptes scabiei* or its ova. In all these cases, however it must be remembered that there may be a coincidental spirochaetal infection, the initial lesion having afforded the necessary route of entry for the organism, and therefore routine serological tests should always be carried out over a period of three months in all cases of genital sore.

A neoplasm is likely to be found in an older age group. It has a typical rolled edge, and the mobility of the lesion over the deeper tissues is likely to be impaired. The duration of a neoplastic lesion is liable to be longer and the lymphatic gland enlargement, which is stony hard, is later in appearing. In a gamma of the genitalia the inguinal glands are not involved, but the serological tests for syphilis are usually positive. There may be other clinical evidence of late syphilis.

(6) **Extra-genital chancre** A chancre of the lip must be differentiated from a gummatous lesion an epithelioma, a primary tuberculous lesion and from herpes simplex with secondary pyogenic infection. Tuberculous ulcers on the tongue carcinomas and aphthous ulcers must be differentiated from a

primary chancre in this position. A similar lesion on the tonsil might resemble Vincent's angina, diphtheria or lymphosarcoma. A chancre of the nipple must be differentiated from Paget's disease. On the finger a chancre is sometimes mistaken for simple paronychia. An epithelioma, sarcoma or anthrax pustule at this site rarely give rise to difficulty in diagnosis. A chancre of the eyelid must be distinguished from a sty. At the anal margin a primary chancre may simulate a fissure, a thrombosed



FIG. 110.

Early Syphilis. Primary stage. Chancre on Lip.

external pile or Bowen's disease. The final diagnosis will rest on the pathological findings. A particular difficulty arises in the diagnosis of a chancre in the mouth or pharynx in that saprophytes may be seen in the exudate which are common mouth saprophytes (such as the *S. Microdentium*) and be mistaken for the *Treponema pallidum*. Gland puncture from the regional lymph nodes may demonstrate the organism by dark ground examination. If these examinations are negative the serological tests should be frequently repeated until the diagnosis is confirmed by positive reactions. The early examination of the patient's sex contacts may be of great value as an aid to diagnosis.

### Secondary Stage

The interval between the appearance of the primary chancre and the secondary manifestations is from six to eight weeks. It is found that over three quarters of patients seen in this stage have skin lesions, about one half have generalised adenopathy about one third have lesions of the mouth and throat while less than one tenth have lesions involving the osseous system, the nervous system the eye or the abdominal viscera.

Cutaneous lesions are varied, and may resemble any generalised skin condition which is not vesicular or bullous. Several types of rash may be present at the same time (pleomorphism). Lesions are non irritating, indurated (except in the macular rash), of a brownish pink (ham) colour and are distributed more markedly on flexor surfaces. They also are indolent, and may persist for weeks or months if untreated, but in some cases they are probably extremely transient as a high percentage of patients diagnosed in the later stages of syphilis give no history of having noted any secondary signs. The rash may only be apparent as part of the Herxheimer reaction, which occurs 6-12 hours after the beginning of treatment with penicillin.

The symptoms of secondary syphilis include sore throat, hoarseness, malaise headache fever arthralgia and bone pains, in conjunction or separately. Clinical features include enlargement of lymph glands, particularly of the posterior cervical, occipital and epitrochlear groups, lesions of mucocutaneous junctions, visceral involvement (liver and spleen) and iritis. The healing primary chancre with local adenitis may still be present, but in twenty five per cent of cases there is no evidence of a primary lesion in the secondary stage.

Positive serological tests for syphilis are found in all cases. The presence of a recent genital sore with regional adenitis is of diagnostic importance. The *Treponema pallidum* may be found on the surface of mucous patches and condylomata lata by dark ground illumination, being identified in ninety per cent of such lesions. It is also present in the serum produced by excoriating papular eruptions of the skin.

The individual lesions seen in any case may be macules, papules, pustules, follicles, annular lesions or scars, and pigmentation may occur. It must again be emphasised that bullous



FIG. 111

Early syphilis. Secondary stage. Macula rash.

and vesicular lesions never occur in acquired syphilis. (Bullous lesions of syphilitic pemphigus occur in early congenital syphilis.)

**Macular (roseolar) rash.** This is the earliest type of skin



FIG. 112.

Early syphilis. Secondary stage. Macul. Papular rash.

lesion to appear. Lesions vary in size in different cases and are of a faint pink colour. They are often not clearly demarcated, but fade into the surrounding skin the colour at times being so delicate as to be invisible in artificial light. Lesions occur chiefly on the shoulders, chest, back, abdomen and upper arms. They may only persist for a few days and may be sparsely distributed.

**Papular rash.** This is composed of pinkish brown (copper coloured) papules varying in size from a quarter to one inch in diameter. Lesions of the smaller size are more common. On palpation the characteristic induration will be recognised. The papules are distributed generally over the trunk, arms and legs,



FIG. 112.

Early Syphilis. Secondary stage. Palmar lesions.

and occur typically on the palms, and soles and on the face. The hair line of the forehead is sometimes a site for a grouping of the papules and is known as the corona venerea. Papules may be scaly and are then termed papulo-squamous or psoriasis.

form, according to the degree of scaling. Macular and papular elements often occur together in which case the rash is termed "maculo-papular."



FIG. 114

Early Syphilis. Secondary stage. Papulo-Squamous lesions on Face.



Follicular rash. This is an uncommon type except on the scalp where it causes alopecia. The hair falls out leaving bald patches of irregular outline, occurring chiefly at the sides and

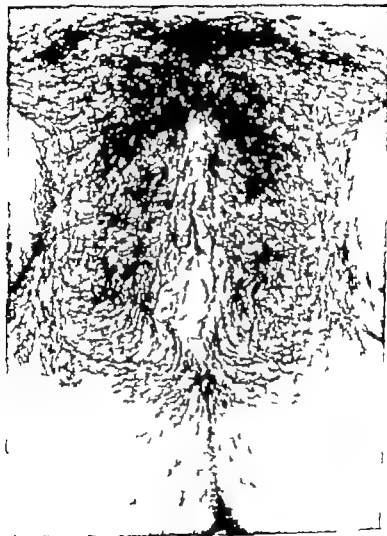


FIG. 115

Early Syphilis    Secondary stage    Perineal and Vulval Condylomata.

back of the head. This gives rise to a "moth-eaten" appearance, in which patches are never well-defined, or completely denuded of hair. In women on account of their long hair it is more difficult to demonstrate this appearance.

**Pustular rash.** This occurs commonly on the face and scalp but occasionally follows a general varioliform distribution. Pustular lesions may produce considerable tissue destruction with overlying layers of crusting producing a "husk" appearance. These lesions are then described as rupial.

**Annular rash.** An annular syphilide is occasionally seen on the face or on the genitalia. It has a higher incidence in coloured races.

**Scars and pigmentation of the skin.** The majority of secondary skin lesions heal without leaving scars. As the papules disappear they leave faint pigmented stains which fade gradually but atrophic macules may persist especially in dark-skinned races.

***Byphilitic leucoderma*** is not a true depigmentation of the skin the real change is an increase of pigment in the skin surrounding the areas of apparent decolouration. It must be emphasised that this is originally a secondary type manifestation which persists for life and therefore is also seen in patients presenting with late syphilis. It affects the back and sides of the neck, and occasionally the shoulders and anterior axillary folds. In the secondary stage it is always associated with a papular syphilide and is more common in dark haired women.

#### ***Lesions of Mucous Membrane and Mucocutaneous Junctions***

(a) **Condylomata lata.** These lesions appear at the anal mucocutaneous junction and sometimes on moist areas such as the female genitalia, the axillae the under surface of the breast or between the toes. This type of lesion is often seen independently of a general skin rash. The lesions begin as papules but on account of the moisture present become hypertrophic with maceration of the surface epithelium. Lesions may fuse together to form plaques.

(b) **Mucous patches.** These occur on the mucous membranes they may therefore be found in any part of the buccal cavity on the tongue the naso-pharynx or the larynx and on the labia

minor in the female. They appear as slightly raised patches, seldom less than five millimetres in diameter covered with a greyish sodden membrane which can be scraped off leaving a pink smooth base that does not bleed. The shape of individual



FIG. 116.

Early Syphilis Secondary stage Macous patches on lips and under Tongue.

lesions is somewhat irregular round or oval, or long and narrow the latter variety being described as "snail track" ulcers. The latter are seen chiefly on the tonsils or pharynx. On the dorsum of the tongue the membrane is often scraped off by constant

infection, and the lesions appear as red patches, denuded of papillae.

Occasionally secondary syphilis gives rise to a diffuse pharyngitis, the mucous membrane being red and slightly oedematous; there is quite often a similar condition of the larynx producing



FIG. 117

Early syphilis. Secondary stage. Follicular lesions of Scalp (Alopecia).

minor in the female. They appear as slightly raised patches, seldom less than five millimetres in diameter covered with a greyish sodden membrane which can be scraped off leaving a pink smooth base that does not bleed. The shape of individual



FIG. 116.

Early Syphilis. Secondary stage. Macrom patches on lips and under Tongue.

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stone type), pemphigus aphtherae (Behcet's syndrome) lichen planus, geographical tongue tuberculous fissures and erosions, and beryth stomatitis. Secondary type lesions on the genitalia may resemble herpes simplex, lichen planus psoriasis and in the female, acute vulval ulcers (Behcet's syndrome)

**Relapse in early syphilis** After the early lesions have healed in response to treatment in the first two years of the disease there is always danger of relapse especially when treatment has been inadequate. As would be expected, the earlier treatment is begun and the more thorough its form the less chance there is that relapse will occur. It may manifest itself by the appearance of signs (clinical relapse) or may be diagnosed by positive serological tests (serological relapse). Thus serum reactions which have been negative for some months may become positive again though the patient has, as yet no signs of clinical relapse.

Relapsing secondary type lesions occur on the skin in the mouth and in the perianal region. In addition to the type of rash already described corymbous, rupial and framboeiform lesions may occur but are now rarely seen. Another form of relapse is occasionally seen, the lesion occurring at the site of the original chancre which it closely resembles, and is termed a "mono-récidive."

The above mentioned are the two common forms of relapse other evidence of treatment failure may include ocular lesions, bone and visceral lesions neuro-recurrence, and finally the birth of a syphilitic child to an apparently cured female patient.

Relapsing muco-cutaneous lesions are highly infectious, it is important therefore to examine patients carefully after treatment in addition to performing the routine serological tests.

## LATE SYPHILIS OF THE SKIN AND MUCOUS MEMBRANES

### Synonym Tertiary Syphilis

Many patients presenting with lesions of late syphilis give a history of the primary or secondary stage of the disease. In

*Treponema pallidum* is not found in the skin lesions, so that the condition is not contagious.

The pathological changes are due to gummatous infiltration.



FIG. 118.

Lat. Syphilis. Not in cutaneous lesions. Not early. Plaque on chan.

The lesions may be classified as cutaneous gummata and subcutaneous gummata. They may appear seven or more years after the original infection in an untreated case. At this stage the only remnants to be seen of lesions of the secondary stage



FIG. 119

Late Syphilis. Ulcerated lesions of Thigh.



are the atrophic macules which may occasionally result from papular secondary rash or the syphilitic leucoderma of the neck.

**Cutaneous lesions** These appear as scaly plaques or groups of nodules, formed of miliary gummata of the dermis which may or may not ulcerate (nodulo-cutaneous or ulcerative types). In contrast to the secondary stage when the lesions are symmetrical, late lesions occur singly or in small isolated groups, being in no way symmetrically arranged. They may occur on any part of the skin surface. The lesions are painless and develop slowly. They display a typical polycyclic border with peripheral hyperpigmentation and the induration of the dermis is easily palpable. There is a tendency to spread peripherally while healing occurs in the centre, finally leaving atrophic "thru paper" scars, which are non-contractile.

**Subcutaneous lesions** A single large gumma of the subcutaneous tissues may present on the skin surface as a smooth painless swelling which occasionally involutes. more commonly it breaks down with discolouration of the overlying skin and discharges its contents. It ultimately results in a deep "punched out" ulcer with a sloughing "wash leather" base. These lesions occur commonly on the upper part of the leg over the scalp and face and in the region of the sterno-clavicular joints.

Late lesions affecting the palms and soles are often symmetrical, although this is not always the case. Ulceration does not occur and the lesions are hyperkeratotic scaly reddish brown patches, with the typical polycyclic outline and indurated edge. Perforating ulcer of the sole (mal perforans) is due to trophic changes and is associated with tabes dorsalis.

**Mucous membrane lesions** Gummata lesions may also involve mucous membranes with destruction of deeper tissues, including bone.

Chronic glossitis is a condition which is commonly observed in late syphilis, there are however other possible aetiological factors, such as smoking and dental sepsis. It produces fissuring and distortion of the tongue together with smooth red areas of denuded epithelium and other areas of thickened white epithelium (leucoplakia). These surface changes are produced by

an underlying interstitial fibrosis. Chronic glossitis produces increased sensitivity of the tongue, which becomes intolerant of strong tastes and spices. This same condition with leucoplakia



FIG. 130.

Late Syphilis. Single Subcutaneous Gumbo of Scalp. Note early central breakdown.

may also occur on the mucous membrane covering the inside of the cheeks, at the angles of the mouth, and on the inner surface of the lips. A single large gumma may occasionally appear as a nodular swelling on the tongue.

These lesions of the mucous membranes may be superseded by

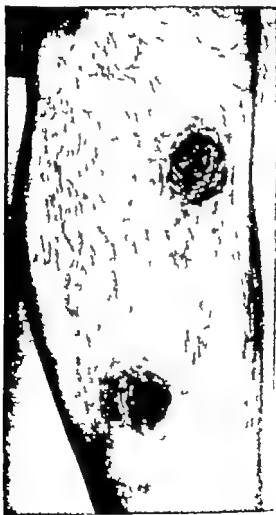


FIG. 11

Late syphilis. b. cutaneous Gummata on Leg

carcinomatous changes, and must be observed with this possibility in mind this is particularly the case on the tongue. A neoplasm may be differentiated from a gummatous ulcer by its



FIG. 122.

Late Syphilis. Chronic Glossitis with marked Leucoplakia.

lack of response to anti-syphilitic treatment, and in the later stages deep infiltration and regional lymph gland enlargement will occur. A biopsy taken at the earliest opportunity will confirm the diagnosis.

**DIFFERENTIAL DIAGNOSIS.** In the diagnosis of late syphilis of skin and mucous membranes the serological tests are positive



FIG. 123

Late syphilis. Chronic Glossitis with Squamous Carcinoma

in about ninety per cent of cases, but a negative result does not rule out the possibility of this diagnosis. In those cases the new specific tests for syphilis such as the Treponemal Immobilisation test (TPI test) will be positive and thus confirm the clinical impression. A previous history suggestive of a genital sore or of lesions of the secondary stage or of early syphilis inadequately treated may also aid in diagnosis. In addition there may be clinical evidence of late syphilis of other systems. Examination of the cerebro-spinal fluid and X-ray and screening of the aorta should be done in all cases before treatment.

The late skin lesions of syphilis must be differentiated from other granulomatous conditions including tuberculosis, leprosy, sporotrichosis, and blastomycosis and various non-granulomatous lesions including the plaques of psoriasis and seborrhoeic dermatitis squamous cell epithelioma of the skin various dermatophytes Hodgkin's disease mycosis fungoides and some types of bromide and iodide eruptions.

The differential diagnosis varies to some extent according to the anatomical site. On the face lupus vulgaris, epithelioma, rodent ulcer leprosy and rhinophyma must be considered. Palmar lesions to be differentiated include chronic eczema, psoriasis fungus infection, papulo-necrotic tuberculide and granuloma annulare. While on the legs stasis ulcers, erythema induratum and sporotrichosis must be excluded.

Tuberculides spread more slowly than the lesions of late syphilis, and are more painful. They affect a younger age group. The scar formation differs, in that it is contractile compared with the tissue paper areas of healed late syphilis of the skin. Stasis ulcers which are of common incidence affect the lower part of the leg and are often associated with varicose veins and a stained eczematous skin condition with pigmentation round the ankles.

### EARLY CONGENITAL SYPHILIS

Skin lesions in children born with congenital syphilis may appear within a few days after birth, or later when the infant is

several weeks old. The rash is part of a generalised tissue reaction to the spirochaetes as a result of a blood stream infection transmitted from the mother to the foetus in utero via the placenta. The early type of eruption "syphilitic pemphigus" consists of groups of bullae, distributed symmetrically on the hands and feet, and occasionally on other parts of the body. This is the only type of syphilitic skin lesion which is ever bullous in nature. The blisters are two to three centimetres in

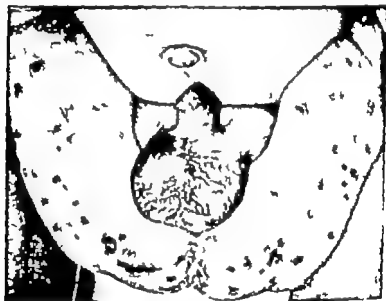


FIG. 124.

Early Congenital Syphilis. Typical Skin and Perianal lesions on Six weeks old Infant.

diameter circular in outline containing sero-purulent fluid swarming with spirochaetes which may be identified by dark ground examination. They have a dark red base and sometimes a brown halo. The prognosis without treatment is grave.

Eruptions which delay their appearance for a few weeks present in a different form. The baby may be born apparently normal, but soon begins to develop symptoms such as nasal

"anuffles" with a hoarse cry and marked loss of weight. At this stage, the skin is never involved alone. The rash consists of small papules on a red erythematous base. The distribution may be general, but is more likely to pick out the buttocks, the palms of the hands, and the soles of the feet, there may be some scaling of the lesions if they have been present for a few weeks. Around the mouth and nose fissures and shallow linear ulcers appear radiating outwards. When these eventually heal they leave deep longitudinal scars which remain for life, and are known as rhagades. Perianal condylomata lata may also be present at this stage, appearing as heaped up masses of greyish epithelium round the anus. Infants in this stage of syphilis are highly contagious.

**DIFFERENTIAL DIAGNOSIS.** The early bullous rash must be distinguished from bullous impetigo (*Staphylococcal pemphigus*). Impetigo does not usually pick out the extremities, and shows no tendency to symmetrical distribution. The papular rash if confined to the buttocks, resembles a far more common condition, a simple napkin eruption. The napkin rash will be most pronounced on the exposed surfaces, where the maximum friction takes place a syphilitic rash on the other hand is seen most clearly in the creases and round the anal orifice. The palms and soles should also be examined carefully if they are affected the diagnosis of syphilis would be supported. Some at least of the associated signs and symptoms are likely to be present and X ray of the long bones and skull may reveal osteo-chondritis. The serological tests will be positive and the *Treponema pallidum* identified by dark ground examination of the lesions.

### LATE CONGENITAL SYPHILIS

Skin lesions in late congenital syphilis are clinically similar to those in late acquired syphilis. Mucous membrane lesions with destruction of deeper tissues including bone, are common. Perforation of the palate and nasal septum are examples of this process. Gummatous lesions occurring in patients below the age of thirty



should always cause the clinician to investigate the possibility of the disease being congenital in origin. A careful history taking including a review of the family history and a search for the typical stigmata of congenital syphilis will usually resolve the problem.

## CHAPTER XII

### DISEASES DUE TO OBSTRUCTION OF VESSELS ARTERIES

**Acrocyanosis** (p 76) is due, according to Lewis, to a spasm of the arterioles of the extremities produced by cold, leading to a slowing of the circulation through the minute vessels of the skin. The minute vessels are at the same time dilated owing to the influence of cold (Haxthausen) and partly owing to the slowing of the circulation through them (Lewis)

**Raynaud's Disease** This is a condition in which the fingers and toes become cold, white or blue and painful, and later red, hot, swollen, and painful. The prolonged interference with the circulation may lead to superficial gangrene. The condition appears to be due (Lewis) to spasm of the digital arteries produced by cold in susceptible persons. It may be followed after some months or years by sclerodactyly. As Raynaud's disease is fully dealt with in text-books of medicine it will not be further considered here.

Gangrene of the fingers and toes may result from obstruction of the arteries by arteriosclerosis, either the general type or the local form which occurs in the toes of diabetics, or by endarteritis obliterans. Also from contraction of the arteries in ergotism and Raynaud's disease. These matters are dealt with in text books of medicine and surgery

### VARICOSE VEINS

**PATHOGENESIS** The veins in the leg consist of two functionally distinct groups, the superficial, which drain the skin and subcutaneous tissues, and the deep. The latter lie together with the muscles, within a tight fascial envelope the fascia lata.

## 274 DISEASES DUE TO OBSTRUCTION OF VESSELS

Certain veins perforate this envelope to connect the two systems. They are the long and short saphenous, joining the deep veins at the *groin* and *popliteal fossae* respectively and certain direct perforating veins, one in the lower third of the thigh and one on the outer and two on the inner aspect of the calf. When the muscles contract, blood in the deep veins is forced upwards, while that in the superficial veins is sucked into the deep system via the perforators. The pressure in the deep veins rising to about 70 mm. of mercury at the inguinal ligament and about 100 mm. of mercury at the ankle. Should the valves in the perforating veins become incompetent, these pressures are transmitted back into them and they react in time by becoming dilated and tortuous, i.e. varicose. When this venous hypertension reaches the capillaries, they can no longer function efficiently the result being the escape of blood and fluid into the tissues, leading to pigmentation and oedema, and malnutrition of the subcutaneous tissues and skin leading to panniculitis, eczema and ulceration.

**ÆTIOLOGY** Varicose veins are between two and three times as common in women as in men. About 80% result from previous venous thrombosis, which damages the valves. The commonest causes of thrombosis are child birth recumbency due to medical or surgical reasons and injury to the leg. In half the non thrombotic group the valvular incompetence is spontaneous and familial occurring in early adult life (primary varicose veins).

**Varicose eczema** A very common condition in middle-aged women and elderly men with varicose veins. The skin on the lower third or half of the inner side of one or both legs has usually been for a long time congested and purplish or else pigmented with blood pigment and scaly. Then it becomes irritable develops vesicles and becomes eczematous. This form of eczema is usually on the inner side but may be on the outer side of the leg depending on which veins are varicose. The condition is very chronic and after it has been present for some months general sensitisation of the skin to products of epidermal break-down takes place so that if the patient then scratches his leg vigorously

or applies an irritating ointment, he is very likely to wake up in the morning with eczema also on his face, neck, arms or hands.



FIG 123.  
Varicose eczema.

Varicose eczema may also arise in the skin overlying a varicose vein in any part of the lower limb.

**Varicose panniculitis** This results from malnutrition of the subcutaneous tissues. It usually occurs in the fat leg but may accompany varicose eczema. A hard, indurated, painful plaque appears, usually above and behind the internal malleolus, but it may be on the outer aspect or even higher up the calf. In the early stages it is red, hot and tender but it steadily progresses to sclerosis of the skin and fibrosis of the underlying tissues, the final appearance being that of a leg which suddenly tapers in its lower third.

**Varicose ulcers** are the most advanced expression of tissue malnutrition. They may accompany either varicose eczema or panniculitis or may appear alone. In the majority of cases they are precipitated by mild trauma. They may have an ovoid or polycyclic outline, the edge may be abrupt or overhanging and is usually indurated. The base is red or purplish and oozes serum, sometimes in great quantity. In area they vary from half a square inch to twenty, thirty or even a hundred and eighty square inches (Dickson Wright). These large ulcers may completely encircle the leg. Periostitis of the leg bones is not uncommon under chronic ulcers and is often more extensive than the ulcer. Varicose ulcers are often very painful.

**DIAGNOSIS** is not usually difficult. Gummatous ulcers (p. 264) and Bazin's disease (p. 214) are the most likely conditions to cause difficulty. The presence of varicose veins, oedema, purple discolouration, pigmentation and thickening of the skin, ulceration on the inner side of the lower third of the leg and extreme chronicity are in favour of varicose ulcer. Syphilitic ulcers may occur in patients who already have varicose dermatitis, but they are likely to be on the outer side of the leg, to begin as swellings which break down in the centre to have a "punched out" appearance, a yellow sloughy base and to be arranged in arcs of circles. The Wassermann reaction will probably be positive but a patient with a varicose ulcer may also have syphilis and so give a positive Wassermann.

Bazin's disease is likely to occur in younger subjects, to be worse in the winter, to begin as subcutaneous nodules which later reach the surface and may then break down, looking not unlike gum

matous ulcers. The lesions of Bazin's disease are usually multiple, some of them at least being situated on the back of the lower half of the leg.

**TREATMENT** The fundamental treatment of varicose veins, and hence of their cutaneous complications, lies in the abolition of the superficial venous hypertension. Ideally this should be done



FIG. 120.

Varicose ulcer. Eight years duration.

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by surgical means by tying off the incompetent perforator veins at their junction with the deep veins and removing by stripping the varicose veins. Frequently however surgical control of the incompetent perforators is not possible and the hypertension being transmitted from the deep to the superficial venous system must be abolished by compression from without. This is done by either elastic stockings or bandages. Both should be as tight as is consonant with comfort and should be worn for the rest of the patient's life. They should be put on each day before the patient gets out of bed and after he has raised his leg in the air for 15-30 seconds. The stockings stretch both longitudinally and transversely (two-way stretch) while the bandages only stretch longitudinally. Various thicknesses of both are available the thicker the material the greater the compression obtained. Bandaging commences at the base of the toes and continues, in an overlapping fashion to below the knee. Stockings can extend to below the knee, mid thigh or groin. Additional pressure localised over the area of an ulcer may be obtained by placing a sponge rubber pad over it, the bandage or stocking being worn on top. When severe induration or oedema is present or when an ulcer is particularly intractable deep massage frequently helps (Bjergaard treatment). It consists essentially in deep massage with finger kneading using zinc ointment as a lubricant, round the edges of the ulcer to other areas of induration and to the whole leg below the knee. This is done for ten minutes to each area twice daily by the patient and once or twice weekly by the physiotherapist. A thin layer of zinc ointment is left round the ulcer then twenty layers of gauze previously soaked in 1% aluminium acetate with 3% boric acid in distilled water are applied to the ulcer covered with oiled silk and kept in place by a few turns of cotton bandage. This dressing should be renewed morning and evening. Wool padding is applied behind each malleolus proximal to the clefts of the toes and round the leg at the level of the tuberosity of the tibia. A rubber elastic webbing bandage (sold especially for the purpose) 11 ft long and 3 in. wide is then applied firmly from the clefts of the toes to the tuberosity of the tibia but is worn only during the day.

The patient should walk five miles a day or bicycle a greater distance.

Varicose eczema is treated in the same way as ordinary eczema (p 331) When the condition is severe, an elastic



FIG. 127

*Elephantiasis nostras.*

bandage may aggravate it and under these circumstances the patient should be treated in bed to begin with. Mild degrees of



eczema however respond well to routine application under a compression dressing

Varicose panniculitis requires no local application but responds well to surgery compression or both. When the condition is painful however an initial period in bed may be necessary

Varicose ulcers should be treated locally with bland applications such as wet gauze compresses of Eusol, saline or 10% urea solution. Occasionally when secondary infection is severe especially if with *Ps. procyanea* the appropriate antibiotic may be applied (p. 33). Large or painful ulcers may necessitate a short period in bed. Occasionally skin grafting is required

### LYMPHATICS

Obstruction of the lymphatics such as occurs in filariasis leads to enormous swelling of the affected parts e.g. elephantiasis arabum and lymph scrotum seen in the tropics. In this country a milder form elephantiasis nostras occurs due to recurrent streptococcal infection. This usually affects the legs. When it occurs on the lips or cheeks it is a sequel to recurrent attacks of erysipelas or recurrent cellulitis (p. 190)

## CHAPTER XIII

### NEURODERMATOSES

Pruritus means itching, and should not be confused with prurigo which is the name given to certain itching diseases.

Pruritus may be general or local. Many skin diseases are characterised by itching, notably scabies, pediculosis, eczema, dermatitis, urticaria, dermatitis herpetiformis, lichen planus, lichen urticatus, mycosis fungoides and prurigo but the term pruritus is used to signify conditions in which itching is the main feature. It may be present with no visible skin lesions, or only those produced by scratching. Different patients vary much in their tendency to pruritus, neurotic and highly strung patients being particularly susceptible. In some cases pruritus appears to be purely psychical. In most cases of pruritus whether general or local, the itching comes on in spasms which may be very severe and lead to uncontrollable scratching. Probably the nerve endings concerned are those normally responsible for pain and for touch.

Generalised pruritus apart from parasites and skin diseases, may be due to external causes such as heat or cold, excessive use of soap or bath salts, irritating underclothing, and very numerous chemical substances dusts etc. (Chap. VI., p. 89) It may be produced by causes internal to the skin such as certain foods, intestinal parasites or alimentary disorders. It may be due to diabetes, or at least to a reduced sugar tolerance to nephritis, lymphadenoma or leukaemia. Diseases of the liver are often accompanied by pruritus which may be severe even in the absence of jaundice. Of diseases of the nervous system it is more likely to be present with functional than with organic disease although it may be an early sign of tabes dorsalis. Cocaine addicts often complain of "insects" or "worms crawling about in the skin and produce as evidence pieces of

epidermis they have scratched off, or little pieces of black thread or other foreign bodies. Pregnancy and ovarian tumours may cause pruritus in women.

Senile pruritus is found in elderly people in whom the skin is dry and atrophic. An attack is often brought on by the exposure of the skin to cold air when undressing.

**DIAGNOSIS.** The first step in dealing with any case of generalised pruritus is to exclude scabies (p. 132). Through neglect of this precaution many cases of scabies are missed. Having done this, lesions suggestive of any other skin disease must be carefully searched for and the underclothing should be examined to exclude pediculosis corporis (p. 143). The history must be gone into to ascertain whether any cause can be deduced from the circumstances of onset or the times at which paroxysms of itching occur e.g. heat, cold foods, drugs (opium, cocaine, hypnotics). If nothing is found a thorough physical examination must be made including such pathological tests as a complete blood examination, sugar tolerance tests and examination of urine and stools. Gout, chronic interstitial nephritis, diabetes, cholecystitis and other diseases of the liver may all be accompanied by pruritus.

**TREATMENT.** If no cause can be found or until the examinations are completed relief must be attempted by the external application of antipruritic drugs. Such are lead and calamine lotions or liniments, with or without the addition of Liq. picis carb. 12% acid. carbol. 2% menthol 2% 5% chloral hydrate 2% chloretone 2% e.g.

B. Calaminae prep	5 i.	12
Zinci oxidi	5 i.	12
Liq. plumbi subacet. dil	5 i.	12
Liq. picis carb.	5 i.	12
Glycerinae	5 i.	12
Aq. calca. ad	5 i.	100

or if the skin is dry

R	Zinc oxide	℥ i.	12
	Lanoline	℥ i.	12
	Aa. Carbolic	℥ v	1
	Menthol	gr v	1
	Ol. olivae } aa.	℥ i.	100
	Aq. calch. } ad		

A clean and pleasant lotion is

R	Chlorethane	gr. li.	0-8
	Liq. plumbi subacet. dil.	℥ x.	2
	Aq. colonicensis	℥ ii.	21
	Aq. dest. ad	℥ i	100

A stronger but less clean one is

R	Liq. plumbi subacet. dil.	℥ i	12
	Liq. picis carbonis	℥ i	12
	Glycerini	℥ i	12
	Aquam Camphorae ad	℥ i	100

A useful prescription in obstinate cases is

R	Chloral hydrate	1 part.
	Menthol	1 "
	Thymol	1 "
	Camphor	3 parts.

Certain proprietary preparations are also useful (p. 91) such as Euzer ointment or lotion, or Quotane ointment. Hydrocortisone is also a potent antipruritic but can only be used on localized areas.

Internal administration of drugs is not so useful, but phenobarbitone  $\frac{1}{2}$  - 1 grain (0.03-0.06 gm.) is sometimes helpful. Occasionally systemic steroid therapy has to be employed in severe cases. Oestrin and stilboestrol are occasionally helpful in female cases and methyl testosterone 5-10 mgm. three daily by mouth, in male or both drugs may be given together in either sex. The antihistamines, phenergan, antihistan benadryl etc. (p. 22) are helpful in some cases of generalised pruritus both by mouth and applied externally. Bran and starch baths (p. 36) are often soothing and are preferable to the use of ordinary soap and

water. As regards diet, alcohol strong coffee, and pungent substances should be avoided.

**Localized Pruritus** Pruritus ani. This is a very common condition and may arise from a large number of causes. The skin affected becomes thickened and excoriated from scratching, and frequently a condition of lichenification (p. 287) is set up which perpetuates the itching.

The causes of pruritus ani may be tabulated as follows

Causes inside the anus	Proctitis with leakage of mucus.
	Patulous sphincter with leakage of mucus.
	Piles.
	Polypl.
	Fissure.
	Fistula.
	Threadworms.
	Constipation.
	Diarrhoea.
	Alkaline stools (Whitfield).
	Leakage of paraffin.
Causes outside the anus.	Pediculosis.
	Lack of cleanliness.
	Excessive perspiration
	External piles.
	Condylomata lata.
	Condylomata acuminata
	Tinea of natal cleft.
	Monilia infection of natal cleft
	Psoriasis of natal cleft
	Eczema of natal cleft.
	Seborrhoeic dermatitis of natal cleft
	Lichenification.
	Vaginal discharge keeping anus moist

In some patients certain articles of food and drink, e.g. alcohol in certain forms and strong coffee or condiments, may cause pruritus ani. It is common also after the use of the "broad-spectrum" antibiotics (v. page 33). Very occasionally threadworms can cause the condition in adults. But when all the above causes have been excluded there remains a distressingly

large number of cases, probably the majority which have to be labelled "idiopathic" as no cause can be found. These are probably examples of neurodermatitis (p. 281)

**TREATMENT** The first step is obviously to ascertain the cause if possible, and then to apply the appropriate treatment. In cases where no cause can be found the patient should wash the parts with cold water after defaecation, dry them well, and then apply hydrocortisone ointment or lotion. One per cent hydrocortisone in a greasy base is usually the most effective form, but sometimes a water miscible base is better and a 2½% strength may be necessary. The strength and frequency of application are slowly reduced once the irritation has been controlled. Complete relief is obtained in about half the cases and occasionally treatment may be discontinued altogether. If hydrocortisone fails to control the condition, the other anti-pruritics already mentioned may be used.

Some patients find that greasy applications increase the irritation, whilst others prefer them. If either a grease or a powder is applied in the morning after the bath it assists in the subsequent cleansing of the anus after defaecation. Very often no application is required in the day but only at bedtime or during the night. A dose of phenegan 25 mg. at bedtime is sometimes helpful as is phenobarbitone ½ to 1 gram.

The majority of idiopathic cases can be relieved by X ray treatment, in doses of 50-100r fortnightly but if doses totalling 800r in a period of three or four months have not cured the condition X rays should be given up. Painting the perianal skin with Thoram X varnish (1500 c.a. units in 1 c.c. p. 48) is sometimes successful, though the reaction may be uncomfortable for a week or two. Psychiatric treatment can be very helpful in obstinate cases, especially if given by a practitioner with experience of this malady.

**Pruritus vulvae** This is a common and very distressing complaint. The scratching leads to lichenification which perpetuates the itching and often to the inoculation of pus organisms which leads to boils on the labia or pubes. In severe cases the patients become depressed or hysterical. Pruritus is

apt to be most severe and intractable in neurotic patients. The causes of pruritus vulvae are as follows

Monilial vaginitis.	Psoriasis of vulva.
Trichomonas vaginitis.	Lichen planus of vulva.
Vulvar warts.	Lichen sclerosus vel atrophicus of vulva.
Condylomata.	Eczema of vulva.
Glycosuria.	Seborrhoic dermatitis of vulva.
Chemical irritants.	Lichenification of vulva
Threadworms.	Leukoplakia vulvae.
Pediculosa pubis.	Senile vulvitis.
Tinea of vulva.	Psychological.
Interrigo of vulva.	

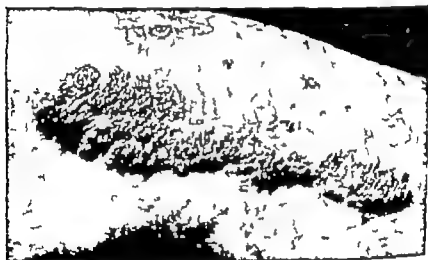


FIG. 128.

Lichenification. Outer side of right thigh. Duration 3 years. Note scars of old boils above

As in pruritus ani in many of the most severe cases no cause can be found and the case has to be put down as "idiopathic"

**TREATMENT** That of the cause if it can be found with the addition of antipruritic ointments and creams as given for

pruritus ani. In many cases with some redness of the vulva but without any obvious trichomonas vaginitis I have found that the use of stavarsol vaginal compound tablets night and morning has had a dramatic effect in curing the vulvitis and irritation. In post menopausal and senile cases oestrogens by mouth are occasionally useful. In cases which appear to have a psychological basis, e.g. unsatisfied desire psychotherapy may help. In idiopathic cases and those with lichenification X rays are very successful, but not more than 400r should be given in three or four months. If this amount fails to cure recourse should be had to painting with thorium X varnish (p. 47).

**Lichenification.** *Lichen simplex chronicus.* *Neurodermatitis.* A condition apt to occur in any area of skin which is constantly rubbed to relieve irritation. The lichenified skin itches and so the vicious circle is complete. It is common on the back of the neck, fronts of the elbows, upper eyelids, inner sides of the thighs and about the vulva and anus. A patch of chronic eczema is very apt to become lichenified and most cases of lichenification seen in the flexures of knees and elbows have developed upon patches of chronic eczema in these situations.

During the Second World War I saw an increasing number of patients with lichenification in multiple areas and have little doubt that this was the result of worry and mental strain due to the war. In some patients these causes had obviously been at work so that neurodermatitis was an appropriate name for the condition.

The affected skin is pigmented and sometimes bluish. It is thickened and the normal cross-cross lines are exaggerated because the islands between them stand up more than normally. Histologically there is a thickening of all layers of the epidermis, exaggeration of the papillae and interpapillary processes and a round cell infiltration about dilated vessels in the corium. The pigment in the epidermis and in the chromatophores in the corium is increased.

**Diagnosis.** From *lichen planus*. This is sometimes difficult but in most cases of lichen planus the typical separate flat topped polygonal, shiny violaceous papules can be found on



some of the typical situations, *e.g.* forearms or penis. Or white spots and streaks may be seen inside the cheeks (p. 364)

**TREATMENT** If the patient can be prevented absolutely from rubbing the patch it generally disappears spontaneously. Sometimes closing it in with an occlusive dressing such as

Viscopaste<sup>™</sup> is effective. Usually the best treatment is by  $\lambda$  rays, 100-200r up to a total not exceeding 800r or by painting with Thorium X (p. 47). These should be assisted by the use of an anti pruritic ointment or lotion (p. 24) and an explanation of the necessity of not rubbing the patch. Podophyllin in a strength of 0.1% in Lassar's paste sometimes causes a cure. The X rays reduce the irritation and the infiltration at the same time and are usually effective. I have seen an area of lichenification vanish by the time the stitches were taken out when a piece had been removed for section. Whether this was the result of complete cessation of rubbing or of some specific effect of the incision I am unable to say.

In cases due to worry and strain the effect of X rays is naturally only temporary and sedatives such as phenobarbitone and amytal with periods of rest and isolation from worries, as far as may be, will be required.

## CHAPTER XIV

### TOXIC ERUPTIONS

These are mostly erythematous and are produced by toxic bodies reaching the skin via the blood stream. The toxins may be derived from foods, drugs (p 118) enemas, or sera (p 129) from the respective organisms causing the acute specific fevers and acute rheumatism, from septic foci from disordered processes in the alimentary canal and from unknown sources.

Generalized erythema is usually bilateral and symmetrical, affecting the trunk, face, and upper parts of the limbs. The eruption may be scarlatiniform morbilliform macular or circinate. Subjective symptoms, itching pricking, etc. are usually slight. General malaise joint pains, diarrhoea, and slight fever  $100^{\circ}$   $101^{\circ}$  F may precede or accompany an attack. The erythema is usually followed by some peeling of the affected skin. The whole attack seldom lasts more than a week or ten days.

**DIAGNOSIS.** From the acute specific fevers. This may be difficult, and often depends upon the mildness of constitutional symptoms, absence of coryza Koplik's spots, adenitis, or other characteristics of the fever in question. In the case of suspected scarlet fever the Dick and Schultz-Charlton reactions may be of service. Pityriasis rosea (p. 333) and secondary syphilis (p 231) must not be forgotten as possibilities in a macular erythema.

**TREATMENT** The cause if discovered must be removed. Failing any discoverable cause the bowels should be kept well open by salines, and the kidneys active by the intake of plenty of water. If any external application is required calamine lotion or a dusting powder is usually sufficient.

Rheumatic erythema is a circinate erythema occasionally seen on the trunk in children with subacute rheumatic fever

*Erythema multiforme*

An eruption consisting of circular or irregular erythematous blotches, bilateral, symmetrical and usually occurring on the backs of the hands and forearms. Other common sites are the dorsa of the feet, legs, knees, sides of neck, face and mucous membrane of the mouth. It has a strong tendency to recur.



FIG. 129

*Erythema multiforme* Forearm.

**ETIOLOGY** The disease can probably be produced by a variety of circulating toxins. It not uncommonly follows vaccination and can be caused by drugs (p 117). At other times it may be produced by toxins absorbed from septic foci, from inflammatory ringworms, from the alimentary canal, and possibly from other viscera. In the majority of cases no adequate cause is discovered. It is more common in women than in men and occurs chiefly in children and young adults.

**PATHOLOGY** The skin vessels are dilated, and there is a cellular infiltration around them. There is more or less serous exudation in the epidermis, sufficient in some cases to cause vesicles or bullae. Occasionally some of the capillaries rupture and the lesions then become purpuric.

**CLASSIFICATION** Descriptive names are sometimes given

PLATE III



Erythema Multiforme



according to the type of lesion present but these appear to have no value for classification. The best division for practical purposes is into (1) cases with maculo-papular lesions, and (2) those with vesicular or bullous lesions.



FIG 130.

*Erythema multiforme. Elbow*

**SIGNS AND SYMPTOMS GENERAL.** There may be either no general symptoms or some malaise with slight fever and joint pains may precede the eruption by a day or two. On the other hand, there may be sore throat, diarrhoea, high temperature and severe pains in some of the larger joints.

**LOCAL. MACULO-PAPULAR TYPE.** The lesions are red or, violetaceous macules or papules on the backs of the hands,

forearms, dorsa of feet, legs, knees and sides of neck. Of these situations the backs of the hands and wrists are by far the commonest. The macules or papules vary in size from one to five centimetres or more in diameter. A common type is that sometimes called erythema *iris* in which circular lesions resembling targets are found on the backs of the hands and wrists. There is perhaps a purpuric spot in the centre of the lesion, and outside this concentric rings of shades of red or violet suggesting the tints of the rainbow (Lat. *iris*). Sensations of burning and tingling may be felt by the patient. The lesions may only last for a few days or may continue to appear for weeks.

**VESICULAR AND BULLOUS TYPE** Here the lesions appear first as red macules, each of which soon develops a vesicle in the centre. The vesicles may remain small or may rapidly enlarge into bullae. In this type the eruption is often extensive and may involve a large part of the body surface. The bullae rupture easily and leave raw surfaces which later become scabbed over. The mucous membranes are often affected in the bullous type and the inside of the mouth may become raw and painful.

**THE STEVENS-JOHNSON SYNDROME** is a name given to a particularly severe type of erythema multiforme involving the skin, respiratory tract and mucous membranes usually those of the eyes, mouth and urethral meatus.

**DIAGNOSIS** Erythema multiforme is not as a rule difficult to diagnose but the vesicular and bullous type may have to be distinguished from pemphigus (p. 446). In erythema multiforme the bullae always arise on pre-existing red macules. In pemphigus they arise on normal skin. Cases with small vesicles may cause confusion with dermatitis herpetiformis. In the latter disease the eruption tends to be less symmetrical, has less tendency to be limited to the extremities, irritates much more, and is often accompanied by an eosinophilia. Cases with the eruption chiefly on the face may be confused with *lupus erythematosus* (p. 303). In this disease however the nose and ears are often affected and the follicles on the affected part are filled with little horny plugs which give the skin a stippled appearance.





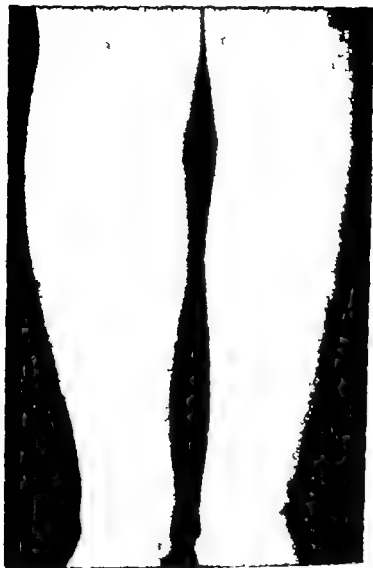


FIG. 131  
Erythema Nodosum.

Most cases occur between the ages of 10 and 30 females are much more often affected than males and the seasonal incidence is mainly in the spring.

**PATHOLOGY** There is dilatation of the vessels in the corium and subcutaneous tissue, inflammation of the arterioles in the subcutaneous fat with localised fat necrosis (Hadfield) exudation of serum and corpuscles and infiltration with round cells and, later giant cells. Capillary haemorrhages may occur



FIG. 122.

*Erythema nodosum.*

**SIGNS AND SYMPTOMS** Acute onset with malaise, fever and joint pains. After one to seven days the characteristic hard, red, painful, and tender swellings appear usually on the front of the legs but also sometimes on the outer sides of the forearms. The swellings are usually oval with indefinite borders, and vary from one to three inches in length. After a day or two they soften and their colour changes like that of a

bruise through shades of violet blue and green to yellow. Each swelling lasts a week to ten days but they may appear in successive crops so that the whole attack may last several weeks. The swellings never break down.

**DIAGNOSIS** From *Bazens disease* (erythema induratum p 214) This is a chronic disease and affects the lower halves of the backs of the legs. It tends to recur every winter. The nodules are purple relatively painless and often break down. From *erythema multiforme* (p 290) In this the lesions are likely to be most marked on the hands or arms and to be maculo-papular or vesicular. They are not painful or tender and there is seldom much fever.

**PROGNOSIS.** The disease usually lasts two to three weeks and the lesions leave no scars. It seldom recurs.

**TREATMENT** Every effort should be made to discover the cause which should then be treated. Further a prolonged follow up should be instituted including twice yearly chest X rays. During the acute phase the patient should be put to bed and treated with aspirin or salicylates. Locally calamine or lead lotion is soothing.

### GRANULOMA ANNULARE

A chronic disease characterised by ringed lesions made up of flat papules or nodules occurring on the backs of the hands or other extensor surfaces. Occasionally the lesions are firm elastic nodules 1-3 cm in diameter situated in the deeper parts of the corium.

**ETIOLOGY** Unknown. Onset may be at any age but the disease is much commoner in children than adults and females are more often affected than males. It lasts for months or years.

**PATHOLOGY** In the corium are nodules composed of lymphocytes, epithelioid cells and fibroblasts with commonly an area of coagulation necrosis in the centre of the nodule. The infiltration is most marked around the sweat glands. There is little change in the epidermis.

**SIGNS AND SYMPTOMS** The lesions are oval or circular from 1 to 3 cms. in diameter made up of smooth infiltrated papules

about  $\frac{1}{2}$  cm. wide, discrete or confluent, and forming rings or plaques. They are usually bilateral though not strictly symmetrical. The colour varies from ivory through normal skin colour to violet. The lesions last for months or years and tend to extend at the periphery and heal in the centre. They very rarely break down usually disappear spontaneously in time without leaving any scar and cause no symptoms except slight itching. The commonest sites are the extensor aspects of the limbs, especially the backs of the hands, but almost any area may be affected.

**DIAGNOSIS.** The appearance is so characteristic that there is little difficulty. An annular lichen planus (p. 366) might resemble granuloma annulare but the lesions would probably be smaller and more numerous and ordinary lichen planus papules would probably be found, if looked for.

**TREATMENT.** Almost any treatment which causes a mild inflammatory reaction may remove granuloma annulare, e.g. rubbing twice daily with a salicylic acid ointment, application of CO<sub>2</sub> snow or the Kromayer lamp. X ray treatment is convenient and usually successful in doses of 200-300r. Occasionally Vitamin E (alpha-tocopherol) is effective in doses of 300-600 units daily. The removal of a small piece, e.g. for biopsy is usually followed by disappearance of the whole lesion. General hygiene should be attended to.

## URTICARIA

### *Nettle-rash*

A condition in which wheals arise on the skin similar to those caused by the sting of a nettle. The wheals arise suddenly and last for a few hours then fade away to reappear later in the same or other places. They are accompanied by severe irritation.

**PATHOLOGY.** The wheals are caused by a dilatation of the capillaries accompanied by an increase in the permeability of their walls, so that serum leaks out and accumulates in the corium. It accumulates to such an extent that the capillaries are compressed by it and the wheal at first red becomes pale except at the edge where it usually remains red. Infiltration



Also as result of trauma, e.g. blow with stick or lash of whip

2. Toxins introduced from without. Stinging hairs of nettles, jelly fish, and woolly-bear caterpillars. Bites of insects and stings of bees, wasps and hornets. Soabica.

3. Toxin reaching skin via the blood stream (a) From the alimentary canal—(1) from some food to which patient is sensitive e.g. shell fish, tinned salmon, stale fish pork, mush rooms, strawberries, and a host of other foods (2) from absorption of some abnormal protein fraction owing to digestive upset (3) from intestinal parasites (4) from certain drugs (p. 117), the most important of which are penicillin and aspirin (5) from the soap used in enemas. (b) From penicillin or serum injections. (c) From the puncture or rupture of hydatid cysts. (d) From some septic focus in teeth tonsils, antra or elsewhere. (e) From *B. coli* infection of urinary tract (Whitfield)

4. Due to emotion, heat or exertion. R. T. Grant and his co-workers have shown (*Clinical Science* 1936, 2, 283) that in six cases of urticaria occurring under these conditions the eruption was due to the abnormal sensitivity of certain cells in the skin to a chemical substance (probably acetylcholine) normally liberated at nerve endings, and that nerve action was the immediate cause of the urticaria.

Urticaria is sometimes an example of allergy and Cranston Low points out that most of the foods which cause it are those which are only eaten occasionally so that the patient has no chance of becoming immunised to them. Ordinary everyday foods, even if they cause an urticaria in childhood, eventually immunise the patient. The same holds with insect bites and stings. Those who are constantly bitten or stung eventually become immune and do not react to the insects attacks.

Urticaria due to cold has been shown in some cases (Harris, Lewis, Vaughan Bernstein) to be due to a dermolyxin circulating in the blood which unites with the skin cells at low temperatures and on rewarming lyses them liberating histamine and so causing urticaria.

In spite of this apparently exhaustive list of possible causes, a great many cases remain unsolved

**CLASSIFICATION ACUTE.** The wheals appear at irregular intervals for several days but gradually become less frequent and eventually cease after a week or ten days. This type is usually due to some food or drug.

**CHRONIC.** The wheals appear usually about the same time of day for weeks, months or years. This is the commonest type seen to-day and it is rare to find a specific cause. Some cases appear to have been caused initially by penicillin but the majority are the result of general stress.

**SIGNS AND SYMPTOMS.** The onset is acute, occasionally with slight fever. The patient notices intense irritation, then a red blotch appears which rapidly develops into a raised white wheal with red edges. Sometimes the process stops short at the red stage and constitutes the so-called "red urticaria," such lesions being little, if at all, raised. The wheals are quite irregular and asymmetrical in distribution. They usually last only a few hours, but may persist for some days. Fresh lesions keep on appearing. The patient scratches, and fresh wheals appear along the scratch marks. In some patients with urticaria, and in some without it, a firm stroke along the skin with say the blunt end of a pencil, causes a rapid red reaction. This is followed in a few minutes by a wheal surrounded by a diffuse bluish or flare (p. 4). This is called *factitious urticaria* or *dermographism*. The lesions so produced appear to differ in some way from spontaneous wheals for they do not itch.

**DIAGNOSIS.** Usually easy. It is important to exclude scabies. Red urticaria may have to be distinguished from *erythema multiforme* (p. 200) but urticaria usually occurs on the trunk and *erythema multiforme* on the limbs. Individual lesions of urticaria also last only for a matter of hours while those of *erythema multiforme* persist for days.

**TREATMENT.** The first step is to endeavour to ascertain the cause, and in the chronic cases this often requires considerable detective ability. Probably many of the chronic cases have their origin in the nervous system and are due to emotion or fatigue. Treatment should be directed appropriately. Most

acute cases are due to some food or drug, and a daily saline aperient with a calamine, lead and carbolic lotion externally for a few days is all that is required.

In the chronic case it is essential to take a very careful history and make a thorough general examination. If the cause is found it must be treated, but in the majority of cases the findings are negative. Skin tests by scarification and inoculation of a number of suspected proteins at different sites along the forearm are generally useless, because the patient usually reacts to the whole lot including the control. Even if one protein does appear to give a greater reaction than the rest if the test is repeated a few days later another protein will probably give the biggest reaction.

One usually has to resort to non-specific desensitization in chronic cases of urticaria. The easiest way to attempt this is to withdraw 10 c.c. of the patient's own blood from a vein in front of the elbow and immediately inject this into the muscles of the upper part of the buttock, midway between the anterior superior spine and the top of the natal cleft. The injections may be made every five days until six have been given.

Sometimes a dramatic cure results after the first injection but often no good effect is achieved. Intramuscular or intravenous injections of calcium gluconate in doses of 5-10 ml daily for a week are also sometimes successful.

Suppressive treatment is best carried out with the antihistamine group of drugs (p 22). Mepyramine maleate ( "Antihistan" ) 100 mg three or four times a day, chlorcyclizine hydrochloride ( "Histamin" ) 50 mg two or three times a day or promethazine hydrochloride ( "Phenergan" ) 25-50 mg at night suffice in the majority of cases. Many other proprietary antihistamines are available however and may be tried. The aim should be to give an adequate coverage over the

24 hours and doses may be increased to the limit of tolerance if necessary. Treatment must be continued for as long as is necessary that is to say until natural remission takes place. In acute cases "Antihistan", "Phenergan" and "Pirton" may be given intramuscularly or intravenously.



Ephedrin gr  $\frac{1}{2}$  (0.03 grm.) 2-3 times a day by mouth or adrenaline 5-10 mg subcutaneously once or twice daily are also useful. In a severe acute attack the latter may be as useful as injections of antihistamines. Sometimes, in suitable cases, gentle sedation with phenobarbitone or amylobarbitone produces dramatic results. Finally in certain cases, both acute and chronic, steroid hormones have to be used.

**Angioneurotic oedema, Giant urticaria.** A form of urticaria in which the subcutaneous tissues are involved.

**ETIOLOGY** It is most common in early adult life and may occur in several generations of a family. It is most frequent in neurotic individuals. An attack may be precipitated by cold, worry, fright, insomnia or digestive troubles. It appears to be due to the same types of causes as urticaria but with a nervous element thrown in, and the tissue affected is the subcutis instead of the dermis.

**SIGNS AND SYMPTOMS.** Attacks usually occur in the early hours of the morning. The eruption consists of circumscribed or diffuse swellings 1-3 inches in diameter or much larger. The swellings do not pit on pressure. The skin over them may be of a natural colour or either whiter or redder than normal. It may be cold and waxy or red and hot. Occasionally it is purplish like a bruise. The usual situations are the face, hands, forearms, genitals, and mucous membranes, but the swellings may occur anywhere. In places where the tissues are loose, e.g. eyelids and genitals, the swellings may be very large.

The importance of the disease is that the swellings may be situated in the mouth, tongue or glottis, and so may cause death by suffocation. Ensor has recorded that in one family of 141 persons in seven generations, 49 were affected and 12 died from suffocation. The swellings reach their full size in a few hours and seldom last more than twenty-four hours. There is, however, a tendency for the attacks to recur indefinitely.

**DIAGNOSIS** From recurrent cellulitis (chronic erysipelas). In this the swellings are of much longer duration (several days), and some permanent thickening of the tissues is left (p. 100).

**TREATMENT** A cause should be searched for as in ordinary urticaria, and the treatment is on the same lines. Injections of adrenalin five to ten minims, or of an antihistamine (Antistin" mg. 100 "Puriton" mg. 10) should be given early in an attack if the upper air passages are involved, and repeated as requisite. It may be necessary to intubate the larynx or to perform tracheotomy in cases of impending suffocation. Cortisone and ACTH have been used with success.

**Papular urticaria.** Lichen urticatus, strophulus, "gum rash" "heat spots." One of the commonest skin affections occurring in infants and young children. The lesions are red blotches or white wheals with a central papule and sometimes a vesicle on the papule. The wheal fades in an hour or two but the papule remains for several days. It irritates intensely until the top is scratched off when it ceases to itch and becomes covered with a small crust.

**PATHOLOGY** Dilatation of the blood vessels in the papillae with oedema both of the papillae and the overlying epidermis and cellular infiltration in the dermis.

**ETIOLOGY** The disease is so common that probably nearly all infants have it in some degree at some stage of their existence. It is usually seen between the ages of six months and three years but may last into or appear first in, later childhood. A proportion of cases appear to be due to insect bites (human, animal or bird fleas, bed bugs etc.) to which the children are particularly sensitive the rest of the family frequently being unaffected. In the majority however no cause can be found. There is little evidence to suggest that the condition is in any way related to urticaria. On the Continent a proportion of these cases are considered to be early mild stages of Hebra's prurigo.

**SIGNS AND SYMPTOMS** The eruption usually appears in the evening and consists of oval or irregular red blotches  $\frac{1}{2}$ - $\frac{1}{4}$  an inch in diameter each with a central papule. The lesions are usually situated on the covered parts of the body especially the lower part of the back and round the waist, but they often occur on the limbs. The red blotches sometimes develop into wheals and the central papules may be surmounted by vesicles. The

blotches or wheals fade in an hour or two and the papules remain. They irritate intensely until the child scratches off the tops of them, when they cease to itch but ooze serum which dries into crusts. Owing to the scratching they frequently become infected with pyogenic organisms and form the basis for a widespread impetigo or ecthyma.

**DIAGNOSIS.** The condition has to be diagnosed from insect bite which it greatly resembles. The minute central red spot where the insect's proboscis entered is, however, absent. The infected cases have to be diagnosed from scabies. Widespread impetigo or ecthyma in a child should always arouse suspicion of scabies or papular urticaria as an underlying cause. In papular urticaria new lesions generally appear in the evenings and are of the character described while no burrows can be found on the characteristic situations (p. 135). The vesicular types have to be diagnosed from varicella. This diagnosis may be extremely difficult, but usually the absence of fever the method of evolution of the spots, the absence of any spots on the palate and scalp and possibly the prolonged duration of the eruption or a history of previous attacks, will lead to a correct diagnosis. Severe lichen urticatus has even been mistaken for mild small pox during epidemics of the latter but careful examination should prevent this mistake.

Papules sometimes become lichenified from rubbing and may then have to be diagnosed from lichen planus but the method of their development should make the diagnosis clear.

**PROGNOSIS.** Attacks vary in duration from days to weeks or months, but seldom recur after six years of age.

**TREATMENT.** Ten per cent D D T (dicophane) powder sprayed on to the patient and his home and furniture is the treatment of choice in those cases caused by parasites. The rest respond poorly to treatment. Antihistamines are helpful in some but non-specific therapy is often just as effective. I frequently prescribe hyd. cum creta  $\frac{1}{2}$  a grain (0.016-0.03 gm.) twice daily with a calamine and carbolic lotion (2%) for external use. Benadryl as Elixir 30-120m 5-40 mg. on the basis of 2 mg. in the day per 1 lb. of body weight may be given.

In impetiginised cases,

R. Hydrarg. ammon.	gr x.	2
Liq. picis carb.	3 i.	12
Ung. paraffini ad	3 i.	100

is useful as it allays the irritation as well as being antiseptic. Hugh Gordon has found ammonium bromide thrice daily in doses of 2 grains (0.13 gm.) for each year of the child a very successful while Sydney Thomson recommends syrup of calcium lactophosphate 3i (4 c.c.) thrice daily. Chloral hydrate may be required to enable the child and incidentally the mother or nurse to get some sleep. In obstinate cases admission to hospital or sending the child to stay in a different house, e.g. with a relative, is usually successful.

## LUPUS ERYTHEMATOSUS

An inflammatory affection of the skin characterised by redness and scaling followed by atrophic scarring. It is most common on the face and has some tendency to be symmetrical. The common type is chronic and localised to the skin, but occasionally the condition is systemic, when it can be both severe and fatal.

**ETIOLOGY** The cause of lupus erythematosus is unknown. In spite of its name, it is in no way associated with tuberculosis. The majority of cases are made worse by sunlight and occasionally the lesions are precipitated by trauma. Many cases are associated with some degree of perniosis and the more severe ones with Raynaud's phenomenon. Dissemination may follow undue exposure to sunlight injudicious removal of septic foci and treatment with various drugs especially sulphonamides and penicillin. Although the majority of systemic cases arise as such, systematization of cutaneous cases is seen. The disease is commonest in northern climates and occurs in women more frequently than in men. The average age of onset in the systemic cases is 21 and in the cutaneous cases 32.

Because the characteristic lesion in the systemic form is a fibrovascular necrosis of collagenous tissue it has been postulated that the condition is an allergic or hypersensitivity reaction and



FIG 134

Lupus erythematosus. Chronic Localised Type. Note destruction of also hair and retraction of lower eyelids.

should be grouped with such conditions as rheumatic fever and polyarthritis nodosa.

**PATHOLOGY** The epidermal changes consist of hyperkeratosis, with plugging of the hair follicles and sweat ducts; preservation of the granular layer; alternate acanthosis and atrophy of the

PLATE IV



Lupus Erythematosus



prickle-cell layer and liquefaction degeneration of the basal cell layer. These changes are most obvious in the cutaneous cases. The dermis is oedematous and the vessels dilated. A markedly patchy mainly lymphocytic infiltrate occurs, concentrated in the vicinity of the hair follicles and sebaceous glands.



FIG. 133.

Lupus erythematosus. Early rather acute type

The characteristic lesion in the systemic cases, best seen in the more severe forms, is fibrinoid degeneration of the collagen. It occurs in the skin but is more obvious in the internal organs, especially the endocardium and serous membranes, the heart



and skeletal muscle the renal glomeruli the spleen lymph nodes and fat depots. Such lesions, repeatedly recurring in the endocardium lead to a characteristic verrucous endocarditis.

**Cutaneous type** **SIGNS AND SYMPTOMS** The disease usually appears as one or more small red scaly patches on the face, neck or ears. These may or may not be symmetrically arranged. The patches extend very slowly and tend to be red and scaly at the margins and to heal up leaving a thin atrophic scar in the centre. Examination with a lens will usually show on some part of the affected skin the characteristic stippling produced by the filling of the sweat and sebaceous gland openings with little horny plugs. On the scalp the scars produced are very atrophic and depressed, with total destruction of the hair follicles, leading to permanent baldness of the affected patch. On the ears the



*Dr. Adamson C. W.*

FIG. 130.

Lupus erythematosus of Scalp. Not disappearance of follicles in central part of scar

lobes are often destroyed. On the hands the disease usually takes the form of oval patches or rings on the backs of the fingers. The red margins of the lips are fairly often affected the mucous membrane of the inside of the mouth much less frequently.

**DIAGNOSIS.** From *lupus vulgaris* (p. 206). The absence of apple-jelly nodules and of ulceration, the stippled appearance produced by the plugged follicles, the central atrophic scar and the later age of onset should distinguish *lupus erythematosus*. *Lupus*

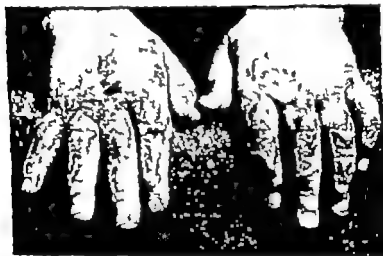


FIG. 137

*Lupus erythematosus of fingers. Ten years duration.*

erythematosus is usually more symmetrical than *lupus vulgaris*, but too much stress can easily be laid on the presence or absence of symmetry in these two diseases.

Slight and early cases may have to be distinguished from *acborrhoea* (p. 390) *psoriasis* (p. 339) *rosacea* (p. 409), *plymoria simplex* (p. 188) or *eczema* (p. 318). On the hands the lesions may have to be distinguished from *chilblains* (p. 75) *erythema multiforme* (p. 290), *papulo-necrotic tuberculides* (p. 216) or *psoriasis* (p. 313). On the scalp from *alopecia areata* (p. 376) and *pseudo-pelade* (p. 374).

**PROGNOSIS.** This should always be guarded, for although many cases clear with treatment others continue to extend for years, or if they heal leave disfiguring scars.

**TREATMENT** The patient should be warned against undue exposure to sunlight. Local treatment is only designed to camouflage and protect the lesions the best application being 2% ichthylol in calamine lotion.

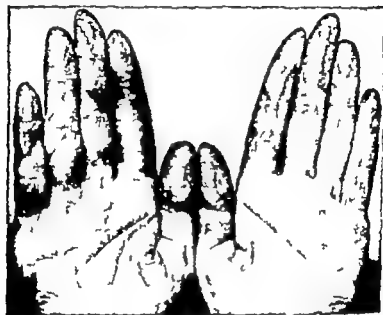


FIG 128

Dr. Johnson &amp; Co., Ltd.

*Lupus erythematosus of Fingers. Note, the dorsal surface is more commonly affected.*

The antimalarial drugs have revolutionised the treatment of cutaneous lupus erythematosus especially chloroquine which is now the drug of choice. It is given by mouth as chloroquine diphosphate or sulphate each 250 mg tablet containing 150 mg of chloroquine base. The starting dose is one tablet twice daily but this may be increased to 5 or 6 tablets a day assuming side-effects are not caused. In about 63 per cent of cases improvement is either complete or very marked response depend



FIG. 126.

Lupus erythematosus, Systemic Type. Patient died of streptococcal septicaemia three weeks after this photograph was taken.

ing on the type of lesion. The superficial, erythematous lesions clear in about 90% of cases, whereas thickened or atrophic patches are much less responsive. Treatment usually has to be continued for at least 3 months and sometimes improvement continues for as long as 6 months. Thereafter further progress is unlikely. Unfortunately about 80% of cases relapse usually in the first few months after stopping treatment, but further courses may be given. Alternately maintenance treatment can be instituted but to be effective the maintenance dose usually has to be as high as the original suppressive dose.

Chloroquine has a number of unpleasant side-effects some of which necessitate reducing the dose or stopping the drug altogether. Occasionally they pass off after the first few weeks. Fractionating the dose and taking it frequently throughout the day especially with meals, also sometimes controls the symptoms. The commonest of these are dyspeptic. A few patients complain of nervous disturbances, such as restlessness, tremors and nightmares. Abnormality of accommodation leading to flickering before the eyes and defective vision occurs in about 5% of individuals and is the commonest reason for stopping treatment. Very occasionally bleaching of the hair and eyebrows occurs.

Mepacrine is seldom used nowadays. It is only about half as effective as chloroquine stains the skin yellow and may cause a severe widespread dermatitis. Very occasionally however a patient may respond to mepacrine and not to chloroquine. The dose is 100 mg. two or three times a day.

Gold (p. 24) has a comparable effect to mepacrine. It is given intramuscularly in the form of myocrisin 0.01 G weekly for 10 weeks being an effective course. Bismuth (p. 23) may also be used the intramuscular injection being given deep into the upper and outer quadrant of the buttock in doses of 0.2 G weekly for 10 weeks.

In spite of all these measures, certain cases fail to respond. In these in which the lesions are usually indurated craggy and atrophic freezing with  $\text{CO}_2$  snow or liquid  $\text{O}_2$  or  $\text{N}_2$  at fortnightly intervals is frequently effective.

**Systemic type** This is usually seen in young women aged about twenty years, and is fortunately rare, for a large proportion of the cases which do occur end fatally. The disease appears to be becoming more common or at least more frequently recognised.

The systemic type of lupus erythematosus may appear as such, at first on the face and later widely distributed or it may arise as an exacerbation of a previously existing eruption of the cutaneous type. The lesions in either case tend to be purpuric and may involve large areas of the body and limbs. They may be mistaken for *erysipelas* or *acute dermatitis*. They are accompanied by arthritis, arthralgia or muscular pains, serous effusions, anaemia leucopenia hyper-globulinaemia with reversal of the albumin-globulin ratio and a false positive Wassermann reaction in 35% high blood sedimentation rate acute glomerulonephritis and a high temperature. Unless adequately treated the disease is generally fatal in a few weeks or months, the commonest cause of death being either uraemia or involvement of the central nervous system. A proportion of the cases show no eruption on the skin and in these, diagnosis must depend on the presence in a young woman, of arthralgia without X ray evidence of bone change fever high erythrocyte sedimentation rate and leucopenia (P O Leary). If there is also a reversal of the albumin-globulin ratio in the plasma, a reduced platelet count, albuminuria and anaemia and if lupus erythematosus cells can be demonstrated the diagnosis is certain. (Orr)

The lupus erythematosus phenomenon leading to the production of lupus erythematosus (L.E.) cells occurs only in systemic cases, especially when severe and when there is a raised gamma-globulin level. It consists in the clustering of polymorphonuclear leucocytes to form rosettes about free, lysed, nuclear material and the formation of L.E. cells which are polymorphonuclear leucocytes each containing a round mass of homogeneous nuclear material staining with haematoxylin. This free nuclear material is the result of disturbance of the nuclear acid metabolism associated with the degeneration of the collagen fibres. The L.E. phenomenon depends on a factor

associated with the plasma  $\gamma$  globulin. It was originally demonstrated by Hargraves in heparinised sternal marrow from cases of disseminated lupus erythematosus but now it is best shown by allowing the patient's peripheral blood to clot (Lee) or by mixing the patient's blood with heparinised normal marrow. Occasionally a few L.E. cells may be found in liver disease.

**TREATMENT** In the milder cases, in which skin and joint manifestations predominate salicylates and chloroquine are the drugs of choice, combined with bed rest. In the more severe cases, steroid hormones must be used but they may be given together with chloroquine. Indefinite maintenance therapy is necessary in about half the cases. Nitrogen mustard has been used for the renal lesions.

## PURPURA

Purpura is the name given to the appearances produced by multiple capillary haemorrhages into the skin. Purpura is a physical sign, not a disease, and it may occur under a large number of conditions. In most of them it tends to be worst on the legs. The immediate cause of purpura is rupture of the capillary walls allowing the escape of blood into the corium. In certain types of purpura this is associated with the presence of micro-organisms circulating in the blood stream and may be due to minute emboli of for example streptococci. In other cases purpura is apparently toxic or is associated with a diminution of blood platelets or of prothrombin or its precursor vitamin K.

**SIGNS AND SYMPTOMS** The lesions of purpura are most commonly petechiae small red or purple macules a millimetre or two in diameter. They do not disappear on pressure. Larger lesions are termed ecchymoses. These may be several centimetres in diameter and resemble bruises. Bullae containing blood may also be found in severe cases. The lesions of purpura appear suddenly and go through the same series of changes in colour as do bruises, viz. red purple greenish and finally yellow.

Purpuras are usually divided into primary and secondary

types, and the classification which follows is that given by Tidy (*slightly modified*)

#### Primary Purpura.

- |  |  |
|--|--|
| 1. Purpura simplex—mild hæmorrhagic type         | } Leakage of whole blood.                          |
| 2. " hæmorrhagica—severe hæmorrhagic type        |  |
| 3. Henoch's purpura—abdominal symptoms prominent | } Leakage mainly of plasma, whole blood in places. |
| 4. Purpura rheumatica—joint symptoms prominent   |  |

For details of these diseases a text book of medicine should be consulted.

#### Secondary and symptomatic purpura.

- |                                |  |
|--------------------------------|--|
| 1. Specific infectious fevers. | Typhus, always.<br>Smallpox, frequently<br>Cerebrospinal meningitis, frequently<br>Scarlet fever in severe cases.<br>Measles, in severe cases.<br>Typhoid fever rarely<br>Other diseases, occasionally |
| 2. Septic infections.          | Infective endocarditis, frequently<br>Septicæmia.<br>Pyæmia.   |
| 3. Blood diseases.             | Leukaemia, especially the acute form.<br>Aplastic anaemia.<br>Thrombocytopenia.<br>Pernicious anaemia, rarely  |
| 4. Toxins.                     | Snake poison.<br>Bera.   |
| 5. Drugs, etc.                 | Chloral.<br>Carbomal.<br>Sedormid.<br>Quinine.<br>Belladonna.<br>Iodides.<br>T.N.T.<br>Neosalvarsan, rarely  |



- |   |   |
|---|---|
| II Constitutional and<br>cachectic conditions.                        | Scurvy<br>Chronic nephritis<br>Disease of liver resulting in lack of<br>prothrombin<br>Carcinoma.<br>Tuberculosis.<br>Old age.  |
| 7 Severe jaundice from any cause with resulting lack of<br>Vitamin K. |   |
| 8 Nervous diseases,<br>rarely   | Tabes, Peripheral neuritis.<br>Hysteria.  |
| 9 Mechanical.   | Venous stasis due to tight bandages<br>Varicose veins.<br>Falling compensation in heart disease<br>On first standing up after long illness.<br>Paroxysms of whooping cough or<br>epileptic attack |

**DIAGNOSIS.** It is important to exclude the severe haemorrhagic types of the acute specific fevers, then scurvy infective endocarditis and blood diseases.

The **PROGNOSIS** and **TREATMENT** naturally depend upon the diagnosis.

**Purpuric dermatitis.** A condition consisting of petechiae haemorrhagic staining and tiny lichenoid papules. It begins usually on the feet or lower legs, but spreads to involve the arms, thighs and trunk. The condition is usually diffuse on the lower legs and forearms but arranged in oval and circular patches on the trunk and upper part of the limbs. Itching is usually severe but may be absent. There is increased capillary fragility demonstrated by gently pinching the skin and showing as linear purpura where the patient has scratched but there is no abnormality of the haemopoietic system and the bleeding and clotting times are normal.

It can be caused by external irritants and ingested substances or can occur for no obvious reason. It thus appears to be a variety of skin reaction analogous to the eczematous reaction. It was first recognised during the last war among troops who

had been issued with new khaki shirts. It was presumably due to some chemical substance left in the material after manufacture, but the exact cause was never found. It was not due to the D D T with which the shirts were impregnated although it has since been definitely caused by that substance. A variety of articles of civilian clothing, all of them new have produced the condition but the commonest cause of all is the ingestion of carbromal (Fig 43). In about 10% of cases, no cause is found.

Treatment consists of finding and removing the cause, when recovery occurs in 2-4 weeks. The ideopathic cases, however may continue for many months. Local antipruritics allay the irritation.

## CHAPTER XV

### ECZEMA

**ECZEMA** has been defined (Goldsmith) as an inflammation of the skin in which the epidermis is predominantly involved, characterised in the early stages clinically by clusters of tiny vesicles and histologically by epidermal spongiosis, that is inter and intra-cellular oedema.

**ETIOLOGY** Eczema may begin in infancy and last throughout life or it may occur in infancy and not later or again it may appear for the first time in adult life or in old age. It is probable that eczema is brought on in most cases by some external irritation, but that often the agent is one which does not cause irritation in a normal person. It follows, therefore, that the subjects of eczema must be abnormal in some respect, and this abnormality of the skin may be hereditary or acquired, temporary or permanent. In other words there appear to be two factors required for the production of eczema a predisposition of the cells of the epidermis and an exciting cause which may be external, blood borne or nervous.

The inherited familial tendency to become sensitised has been called by Coca *atopy* and the associated type of eczema atopic eczema. The sensitisations from which members of atopic families suffer may cause asthma, hay fever eczema, Berrier's prurigo and gastro-intestinal upsets, according to the situation of the sensitised cells. In eczema these are in the epidermis. The allergen to which the cells are sensitised may be derived from outside, from the alimentary canal from bacteria in some septic focus, or from the breakdown of the epidermic cells themselves (Whitfield). It may even be a substance normally liberated at nerve endings as the result of nerve action. This has been shown to be the cause of certain cases of urticaria (p. 299). It has not

yet been proved for eczema, but if true would explain the well-known fact that nervous strain or exhaustion has a pronounced effect in precipitating or aggravating an attack of eczema.

Cases of eczema are not necessarily due to sensitisation. It may be that some people's skins react with eczema to influences, either chemical or physical, which in others would produce a different variety of dermatitis or none at all. In other words in a



FIG. 140

Dr. J. M. M. Case

Eczema. Vesicular and Crusted Type on Forearm.

potentially eczematous subject any external irritation may bring on an attack.

**PATHOLOGY** The first visible stage in eczema is a dilatation of the capillaries, resulting in an erythema. The epidermis and upper layers of the corium then become oedematous the oedema in the epidermis being both inter and intra-cellular the condition known as spongiosis. The inter-cellular oedema puts a strain on the inter-cellular fibrils of the prickle-cell layer and some of these rupture. The cells are then pushed apart and a microscopic vesicle results. According to Percival and Hannay (*Brit. Jour. Dermat. & Syph.* 1919 61 41) actual liquefaction of the epidermic cells takes place on a considerable scale in the formation of eczema vesicles. Such vesicles increase

in size and are pushed up to the surface by the growth of the epidermis. These microscopic vesicles run together until they become of a size visible to the unaided eye. They can then be seen closely set all over the surface of a patch of eczema at a certain stage of its evolution—vesicular eczema. The horny layer over the closely set vesicles gets rubbed off and a weeping surface is left which oozes serum—weeping eczema. After a



*Dr. Adamson's Case*

FIG 14L

*Eczema. Acute Vesicular and Weeping, Patchy Type*

time the serum oozes less rapidly and dries into crusts—crusted eczema. Or the surface may get infected and converted into impetigo (p. 182)—impetiginised eczema. Eventually the horny layer reforms under the crusts but owing to the oedema of the prickle-cell layer the horn cells are not normal and scaling (parakeratosis) results—scaly eczema (p. 5).

Usually the process does not proceed at once to complete cure

but after getting perhaps to the scaly stage the condition relapses and the weeping stage reappears. Alternating improvement and relapse are very characteristic of eczema.

In the corium a small round-cell infiltration about the vessels appears very early in the disease.

In acute attack of eczema such as that described above may after several relapses proceed to complete cure. In many cases however having reached the scaly stage it passes into a chronic state characterized by scaling, thickening and pigmentation of



FIG 143  
Eczema. Discoid Type.  
Front of Thigh.

the skin with exaggeration of the normal ridges and furrows—lichenified eczema. This itches and the patient constantly rubs it, thereby perpetuating the condition. This type is particularly common in the flexures of the knees and elbows. Eczema may however be dry and scaly from the start and may never pass through the weeping stage. Many cases of chronic eczema are of this type.

In the dry scaly and lichenified types of eczema there is pronounced thickening of the horny layer and of the prickle-cell layer with exaggeration of the interpapillary processes

(acanthosis) and a dense cellular infiltration in the corium consisting of small round cells, polymorphs and connective tissue cells around widely dilated vessels.

**CLINICAL FEATURES.** Some of the stages or types of eczema have been mentioned above, viz. *erythematous vesicular weeping crusted dry and scaly impetiginised and lichenified*. In addition there is a form known as *papular eczema* in which the eczematous process is not continuous over the affected area but only occurs at discrete points which are therefore raised up and form papules, often situated about the hair follicles—*follicular eczema*. These may eventually coalesce and form



Dr. Adamson, Lond.

FIG. 143.

Eczema. Follicular Type. Back of Hand and Forearm.

plaques or discs—*discoid eczema*. These discs sometimes heal up in the centre, leaving rings which may be mistaken for ringworm. This type of eczema usually occurs on the extensor surfaces of the limbs. It often becomes lichenified from rubbing and is resistant to treatment.

*Eczema rubrum* is the name given to a very chronic and resistant type of eczema occurring on the leg between the knee and the base of the toes. The surface is red and shining and may weep a little. There may be some crusting especially at the edges, which are well defined. There is much burning and irritation.

Eczema on the *palms and soles* differs from the disease elsewhere because of the great thickness of the horny layer in these regions. The result is that the vesicles rupture with



FIG. 161.  
*Eczema Rubrum.*



difficulty and so generally grow to a diameter of one to two millimetres and persist for a long time looking like grains of boiled sago in the skin. This type constitutes one of the forms of pompholyx (p 336). Chronic scaly eczema on the palms and soles tends to be associated with deep fissures because of the loss of elasticity of the infiltrated skin—*eczema fissuratum*.

Eczema of the distal phalanges often affects the nail matrices, causing the nails to show transverse grooves and irregularities.

Eczema may be localised to a few small spots or patches or



Dr. Adamson, Camb.

FIG. 145

Chronic Eczema of Fingers with Secondary Streptococcal Infection.  
Note transverse furrows on nails representing previous attacks.

may be so widespread as to involve most of the skin surface. Practically all cases of eczema are characterised by itching or burning sensations which are usually worst after washing with soap and water and when the patient is hot or tired. The itching leads to scratching which aggravates the eczema and may lead to infection or lichenification.

Infective eczematoid dermatitis is a form of eczema which arises around a focus of chronic infection such as an infected wound, ulcer or sinus, a chronic discharging ear or an X ray burn

and is due presumably to the skin becoming sensitised to the infecting organisms or to one of them. The eruption may be red, *eczema*, vesicular pustular or crusted or else dry and scaly. The difficulty before the introduction of antibiotics was to eliminate the infection without aggravating the eczema. Now the application of an antiseptic cream or ointment (p. 33) is often rapidly effective, especially if combined with hydrocortisone (p. 38). Alternately one of the quinoline compounds may be used (p. 183), such as Vioform cream, or one of the dyes, for



FIG. 146.

Eczema of Nipples and Areolae. Patient aged 18. Contract with Paget's disease, fig. 901

instance 1% aqueous gentian violet. When the infective element has been dealt with, Lassar's paste or a weak tar paste will complete the cure.

Varicose eczema, see p. 974

Infantile eczema. This usually begins about the age of 6 months and in the majority of cases clears up by about the third year. The exact cause is unknown. A specific allergy to one or more substances, whether ingested or inhaled is not an important factor. For example although some two thirds of all cases give positive skin reactions to one or many foods when introduced by scratch tests, the eczema is not altered in any way whether the foods are included in their diet or not. Clearly there

is an inherited congenital predisposition to the disease in many cases and in the severer cases especially those in which the malady persists into later life this predisposition may amount to an actual anatomical or functional defect. Psychological



FIG. 14

Infective Eczematoid Dermatitis round superficial wound below right knee Duration 1 month.

factors are also important and a disturbed mother-child relationship resulting from the mother's conscious or unconscious hostility to the child is frequently present.

Whatever the essential cause there is no doubt that all cases

of infantile eczema are made worse by exposure to hot fires or hot sun, cold winds or strong soaps, and according to Bamber most cases of infantile eczema begin in the winter months, which suggests that changes of temperature are important. In my own experience most babies with infantile



FIG. 149.

Infantile Eczema.

eczema have been fat and rather overfed looking. In many cases there is a family history of allergic diseases (atopy).

**CLINICAL FEATURES.** Infantile eczema has a characteristic distribution viz. on the flush patches of the cheeks, the forehead and the chin. It may remain limited to these parts or it may extend on to the scalp, wrists, legs and trunk. The affected

skin is red and rough with minute cracks oozing serum or closely-set vesicles on a hot, red, swollen surface. The itching is great and the rubbing and scratching quickly lead to removal of the horny layer and the exposure of a weeping surface. This becomes crusted as the serum dries. The disease varies from time to time, having a great tendency to relapse when nearly well. The child is usually very fretful at night and its mother is often completely worn out from lack of sleep, while the baby apart from the eczema, seems little the worse.

Infantile eczema has to be distinguished from *seborrhoeic dermatitis* in infants. This begins on the scalp with yellowish greasy looking scales on a reddened base and extends down behind the ears and on to the face. It tends to affect the central parts of the face about the mouth and nose more than does eczema proper and may also involve the flexures, axillae, groins, natal cleft and folds about the thighs (p. 306).

Beunier's prurigo is a name given to a condition of eczema and lichenification affecting the face and the flexures of the elbows and knees and associated with asthmatic attacks. More than half the cases have a history of infantile eczema. The following points in Beunier's prurigo were emphasised by Rasch: familial incidence; onset in early childhood (much more rarely at or after puberty); intensity of itching with nightly exacerbation; chronicity and resistance to treatment; coincidence of asthma, attacks of which tend to accompany the paroxysms of itching (or more rarely alternate with them); occurrence in pale, nervous, restless children; distinction from prurigo of Hebra and good effect of a strict milk and vegetable diet.

The disease tends to get less or to disappear between the tenth and the fifteenth year but in some cases it persists throughout life.

About 10% of cases of Beunier's prurigo develop a particular type of ocular cataract the onset occurring usually between the ages of 15 and 40.

**DIAGNOSIS:** Eczema, because it can appear on so many parts of the body and can have so many different appearances may be confused with a very large number of other skin diseases.

My own practice, except in cases which are absolutely typical, is always to consider all other possible diagnoses first and only to diagnose eczema when I am satisfied that I have not missed



FIG. 140

*Bennier* Prurigo showing lichenification in front of elbow

anything else. The diagnostic index at the beginning of this book should be consulted for suggestions as to the most likely alternative diagnoses.

In attempting to discover the cause of eczema in a given case

a "patch test" is sometimes helpful. The suspected substance is kept applied to a square inch or so of skin under strapping for forty-eight hours. A pronounced excretaneous reaction throws further suspicion on the substance tested (Fig. 22). Sensitisation in eczema, however, is often multiple. A control with strapping alone must always be done.

**PROGNOSIS** Eczema can generally be cured but is always liable to relapse or to appear elsewhere. The fact that a patient has once had eczema shows that he is a person who can develop it and that being so he must be regarded as a potential candidate for it whenever the unknown combination of circumstances which caused it at first, arises again. When an intelligent patient has had it several times he may be able to associate the attacks with something which he eats or does, or uses, and so avoid it in future. Some cases of chronic eczema are exceedingly resistant to treatment.

**TREATMENT** *General* The first step is to endeavour to ascertain by minute enquiry whether the "eczema" has been brought on by exposure to any recognisable irritant. If a satisfactory cause can be found then it should be removed and the prognosis will be correspondingly improved. The essential feature is to secure protection of the skin from external irritation and to allay existing inflammation and itching. Having excluded any recognisable external irritant, the patient must be carefully examined to exclude any septic focus, e.g. in tonsils, teeth, appendix etc. Possible psychological causes should also be looked for.

In a case of extensive or severe eczema the patient should be kept in bed not only because rest is valuable but because staying in bed avoids friction from clothes and makes it easier to keep ointments, etc. properly applied. The bowels should be kept open. The diet should be light and simple. Alcohol should be avoided. Medicinal treatment is not of great value except for the hypnotics and steroid hormones. To procure gentle sedation the former may be given through the day in the form of amylbarbitone gr  $\frac{1}{2}$  (mg. 15) or phenobarbitone gr  $\frac{1}{2}$  (mg. 30) two or three times. To insure sleep higher doses must be given and the

barbiturates on the whole are the most effective. Occasionally antihistamines may decrease irritation, especially promethazine hydrochloride ("Phenergan") which because of its added hypnotic effect, is best given at night in 25-50 mg doses. Steroid hormones (p 35) are invaluable in severe widespread and intractable cases, especially in severe exacerbations of Bernier's prurigo, but should be reserved for these.

**LOCAL TREATMENT** Local treatment is by far the most important in eczema. In the first place it should be emphasised that water and especially soapy hard water is irritating to an eczematous skin. Many people have small patches of chronic eczema on the legs and arms which will disappear if they are anointed daily before the patient's bath with Ung. paraffini so as to waterproof the patches and prevent the soapy water from irritating the eczematous skin. Crusts, if present must be removed either by mopping with olive oil or liquid paraffin, or by the application of starch and boracic poultices (p. 28). If the eczema is infected, boracic fomentations should be applied at quarter hourly intervals for an hour or so at a time, or wet dressings of 12% sodium sulphate constantly applied. Alternately local antibiotics may be used (p 33), with or without the addition of hydrocortisone (p 38). If the infected eczema is on the hands or feet the affected part should be soaked in a bath 1/8000 potassium permanganate (enough crystals to make the water a dark pink colour) for five minutes three times a day and then dressed with lead and calamine lotion.

It is essential to prevent the patient from scratching. The best way to do this is to allay the irritation, but in infants restraint may be necessary by tubular cardboard splints round the arms, or by tying the hands and feet to the cot so that they can be moved, but not far enough to scratch any part.

**LOCAL APPLICATIONS** The nature of these must vary according to the type or stage of the eczema present. If the surface is red or vesicular a powder e.g. zinc oxide, calamine, talc or a mixture of these should be dusted on, or a lotion, e.g. lead or calamine or both combined, may be repeatedly mopped on the



part, or else applied on gauze or butter muslin and lightly fastened on. If the surface is weeping lead  $\frac{1}{2}\%$  silver nitrate, or 2% aluminum acetate lotion may be applied in the form of wet gauze compresses three times a day the dressing being re-wetted by pouring on more lotion as soon as it becomes dry. Such wet dressings should not be covered with any waterproof material and should be secured with as little bandage as possible so as to avoid heating the part. The minimum amount of covering should always be used in eczema and dermatitis at any stage. Thick layers of lint or cotton wool are very bad because they cause retention of heat and consequently aggravate the inflammation and itching. On the limbs, where bandages are difficult to keep on, pieces of tubular stockinette bandage or light coloured silk or artificial silk stockings with the feet cut off can be used to keep dressings in position. Or pieces of gauze may be stitched or pinned to the inside of the underclothing at the appropriate points.

When the weeping stage has passed and in less acute types of eczema, calamine linament and cream or zinc paste may be used. The latter should be spread thickly on butter muslin and laid on the part being secured by as little bandage as possible. It should be renewed once or twice daily and before renewal any old paste which has dried up should be gently mopped off with olive oil or liquid paraffin. Old paste which has not dried up should be left *in situ*.

Formulae for the preparations referred to above are

Lead Lotion				Lead and Calamine Lotion			
B	Liq plumbi subacet.	M vi	12	R	Calaminae prep	3i	12
	fort				Zinci oxidi	3i	12
	Aq dest. ad	3i	100		Liq plumbi subacet		
	If this proves too drying milk may be substituted for the distilled water				dil	3i	12
					Glycerinae	3i	12
					Aq calcis ad	3i	100

Calamine Lotion.			Zinc Cream		
R. Calaminae prep.	3 ii	23	R. Zincs oxidi	3 i ii	12 25
Adipis lanae			Adipis lanae hyd	3 i ii	12 25
anhyd.	gr v	1	Ol olivae	} aa. ad 3 l	100
Acid. oleio	℥ iiss	0.5	Aq calcis		
Ol. olivae	} aa. ad 3 l	100			
Aq calcis					

Zinc Paste			White Tar Paste		
R. Zincs oxidi	3 ii	25	R. Picric carb prep.	3 ½ l	} 6-12
Pulv amyli	3 ii	25	(B P C.)		
Adipis lanae			Zincs oxidi	3 ½ l	} Misco. 18-12
hyd.	3 ii	25	Pulv amyli	3 iii	
Paraffin moll			Paraffin. moll	3 iii	} Misco. 38
ad	3 l	100			

Antipruritic substances which may be added to calamine lotion, zinc cream or zinc paste are

Liq picis carb	℥ x 3 l	ad 3 l	2 12 per cent.
Ac carbol.	℥ v ℥ x	ad 3 l	1 2 "
Menthol	gr v	ad 3 l	1 "
Ol. menth. pip.	℥ v	ad 3 l	1 "
Chloretono	gr v	ad 3 l	1 "

Finally in chronic or resistant cases, the addition of iodochlor hydroxy quinoline ("Vioform") or tar to the paste hastens recovery. Vioform cream contains 3% iodochlorhydroxy quinoline and may be added to zinc paste in amounts varying from 10 to 50%. Tar may be prescribed as solution of coal tar (liquor picis carbonis) and in strengths of 2 to 100. is the main constituent of weak tar pastes, a very good one being solution of coal tar 20 ℥ (4%) ac. boris 20 gr (4%) pulv amyli 2 drams (25 o) equal parts of zinc ointment and soft paraffin to 1 ounce. Crude or prepared coal tar is used in strong tar pastes in strengths of 12% e.g. White tar paste.

In resistant patches of eczema painting with prepared coal tar (B P C.) is often very useful. The tar is painted on in the

thinnest possible layer and allowed to dry for ten to fifteen minutes. It is then powdered over with talc powder and if necessary lightly bandaged to protect the patient's clothes. The painting is renewed every second or third day as may be necessary.

Local hydrocortisone in the form of a lotion or ointment (p 38) may be used at any stage of the eczematous reaction and is frequently helpful. It is most effective in the acute and sub-acute stages and should be reserved for localized patches. It is particularly useful in infantile eczema but frequently disappointing in eczema of the hands and feet.

*INFANTILE ECZEMA.* Infantile eczema must be treated on the lines indicated above for eczema in adults. As it is usually on the face it is generally necessary to make a mask of butter muslin with holes for the eyes nose and mouth and after smearing this thickly with zinc paste to secure it over the face and renew it twice daily. Tar preparations are very useful in treating infantile eczema, e.g. prepared coal tar five or ten minims in an ounce of zinc paste. Pot. bromide or chloral or benadryl elixir may be required to allay restlessness.

*X RAY TREATMENT IN ECZEMA.* X rays are of the very greatest value in clearing up subacute or chronic patches of eczema. The irritation is allayed the oozing ceases and the thickening disappears in a few days in a most dramatic manner. Only small doses, e.g. 50r 100r are required. They should be repeated once a week for two three or four times. The intervals may be lengthened to 10 or 14 days after the first two doses. Simple protective treatment by zinc paste or calamine lotion should be continued during X ray treatment, though the zinc should be removed before the actual application of each dose.

*PROTEIN SHOCK TREATMENT* (see Chapter III p 23) is sometimes of value in persistent eczema. Occasionally its effect is dramatic.

*ULTRA VIOLET LIGHT TREATMENT.* Occasionally cases of eczema are benefited by ultra violet light treatment. The dosage must be very small to start with and only slowly increased. The majority of cases are made worse by light.



FIG. 130.  
*Chetropompholyx.*

**PROPHYLAXIS** A patient who has once had eczema should take care to avoid exposing his skin more than is necessary to any irritant which might precipitate an attack. From this point of view hot sunshine, hot fires, cold winds, soap soda (bath salts) and water must be classed as irritants. This does not mean that such a patient should never use soap, but he should only use a superfatted soap and should not rub even this on the areas which are subject to eczema. On places where soap cannot be used at all cold cream may be used for cleaning purposes as suggested above, waterproofing susceptible parts of the skin before the bath with Ung. paraffini or Ung. simplex is a useful measure. In winter such patients must take care not to get their hands chapped and should always grease them before or after washing. An eczema patient should also carefully observe the effects of food and drink upon himself in case he may be able to identify some article which appears to precipitate an attack.

**Pompholyx.** A name given to vesicular eruptions on the hands (cheiropompholyx) or feet (podopompholyx). The majority of such eruptions are merely eczema (p. 321), whose appearance is modified by the thickness of the horny layer in these places. This variety of eczema seems sometimes to be brought on by mental stress. Other eruptions included under the term cheiropompholyx are *dermatitis due to external irritants* (p. 89) and *toxic eruptions* (epidermophytides) on the hands due to ringworm infections between the toes or elsewhere (p. 167). Actual ringworm infections of the skin of the palms causing vesicles are rare in Great Britain although they seem to occur in some other countries. Vesicles due to infection of the soles of the feet with fungi are however common in this country (p. 163).

Owing to the great thickness of the horny layer on the hands and feet eczema vesicles there cannot rupture as they would elsewhere and they remain for many days like grains of boiled sago in the skin. The best treatment is calamine and lead lotion with or without liq. pcis carbonis with, in addition weekly doses of 100r of X rays up to four times. Such vesicles may get infected and become purulent. If infected the hands or feet should be soaked in 1/8000 potassium permanganate solution

for 5 minutes three times a day and then be dressed with strips of gauze dipped in lead and calamine lotion. Large blisters should be opened, and infected ones cut away altogether before soaking. Antibiotic ointments are useful in some cases and in severely infected cases systemic antibiotics may also be necessary.

Another condition in which purulent vesicles occur on the palms and soles is *acrodermatitis perstans*.

*Acrodermatitis perstans* A name given to a group of conditions all characterized by flat, yellow sterile pustules occurring under the horny layer of the epidermis of the palms and soles. The pustules may occur symmetrically on both hands, commonly on the thenar or hypothenar eminences, or on both feet, commonly under the insteps. Or they may appear on one hand or one foot only or on all four extremities. They are accompanied by slight or moderate discomfort or irritation. The pustules dry up turn brown and then exfoliate, while fresh ones continually appear. As its name implies the condition may be very persistent. In a series of 25 of my cases 9 were in men and 16 in women, the average age of onset being 43 in men and 36 in women. Two men, with evidence of proriasis elsewhere, had had *acrodermatitis perstans* for 7 and 13 years respectively and one with evidence of eczema elsewhere had had it on his left foot for 20 years, and right foot for 5 years when first seen.

**DIAGNOSIS.** From *reticular ringworm* which is usually unilateral. By microscopic examination of the roofs of the pustules after warming in liquor potassae (p. 159). Fungus is always easily found in *reticular ringworm*. From a secondarily infected "pompholyx" i.e. eczema or dermatitis. In such cases the pustules are usually much larger and more prominent and there is much more inflammation, pain and disability. Culture from the pustules will grow an infecting organism, usually a staphylococcus. From *dermatitis repens* (Crocker). This is a very rare condition. It usually follows an injury and starts about the nail of one digit, spreading relentlessly to the other digits and the palm or sole. It is usually confined to one extremity and staphylococci capable of growing on media usually inimical to them can be grown from the pus.

The term *acrodermatitis perstans* seems to cover three separate entities.

1. *Pustular psoriasis* Barber and also Ingram as well as my own observations have shown that some of these cases are a pustular form of psoriasis affecting the extremities, the histology of the lesions being that of psoriasis. In some of these patients obvious psoriasis, slight or severe, is present elsewhere. (23% of my series of 25 cases.) (Fig. 161)

2. *Pustular bacterids* (Andrews) In these cases there is no evidence of psoriasis but infective foci can be discovered elsewhere e.g. in tonsils, teeth or sinuses and the lesions clear up only when the infective focus is removed. The histology of these cases does not resemble psoriasis and is characterised by pustules deep in the epidermis with very little, if any surrounding inflammation. Some cases I have seen certainly belong to this group.

3. *Eczeema* I am satisfied in my own mind that there is a third group which should be regarded as a peculiar form of eczema, for the patients have typical patchy vesicular eczema elsewhere. (16% of my series of 25 cases.)

I have found it very difficult in the absence of obvious psoriasis or eczema elsewhere to decide without a biopsy to which of these three groups any given case belongs. At one time I thought that in psoriasis cases the pustules were confined to a sharply limited patch and in the bacterids they were more diffusely scattered over the palm or sole but recent experience does not confirm this.

**TREATMENT** These cases are among the most intractable in dermatology. A septic focus should be excluded or if found dealt with appropriately. As a general rule White's tar paste full or half strength (p. 333) is the best application. It has improved or cleared up 52% of my series of 25 cases, whereas X rays in doses of 100r weekly or fortnightly only improved 33%. A few patients do better with Ung. hyd. ammon. co. (p. 333)

## CHAPTER XVI

### PSORIASIS AND PITYRIASIS ROSEA

#### PSORIASIS

A chronic disease characterised by sharply defined patches of erythema covered by silvery scales.

**ETIOLOGY** Although psoriasis is one of the commonest skin



FIG. 161.

Psoriasis. Common Type Left Knee.

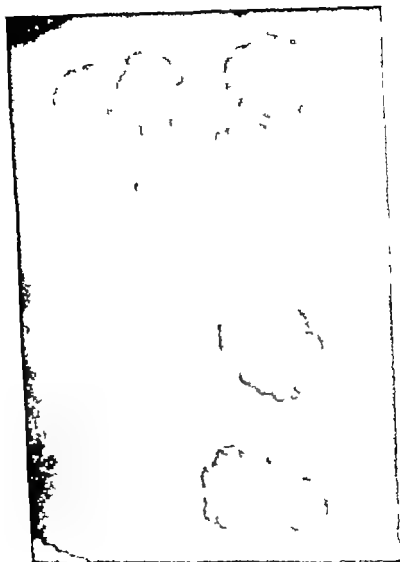
diseases in this country its etiology is completely unknown. It may occur at any age, although it is rare in infancy and old age. The commonest age of onset is five to fifteen years. It is not infectious, and a tendency to it appears to be hereditary





FIG. 18.  
Psoriasis. Guttae Type

PLATE V



Gyrate Psoriasis



in about 30% of cases. The sexes are equally affected. It is commoner in the colder northern countries of Europe than in the southern, and in fair haired than in dark-haired persons. It is rare in the tropics. It tends to appear and to recur in



PLATE 183

Psoriasis. Flexural type on knee, of 20 years duration.

psoriasis in some cases, in autumn in others. Nervous influences may determine an attack e.g. I have had three patients who always got psoriasis when they were preparing for examinations. Trauma often determines the site of a patch of psoriasis, e.g. a scratch. This probably accounts for its commonly affecting the knees and elbows.

**PATHOLOGY** The characteristic changes in psoriasis are

1 In the corium exaggeration of the length of the papillae by down-growth of the interpapillary processes of the epidermis dilatation of the capillary loops, oedema of the papillae, especially of the distal portions of them small round cell and polymorph infiltration. Plasma cells are absent.

2 In the epidermis, acanthosis, i.e. great exaggeration of the thickness of the prickle-cell layer with resulting down-growths of the interpapillary processes. The portions of this layer over the summits of the papillae are however normal or less than normal in thickness. Oedema of the prickle-cell layer with absence of the stratum granulosum and imperfect formation of the horny layer so that the horn cells are nucleated and stick together forming scales (parakeratosis). The silvery appearance of the latter is due to included air. "Microabscesses" or "dry abscesses" in the thickness of the epidermis. These are small collections of mononuclear and polymorphonuclear cells which have migrated from the vessels at the summits of the papillae and have travelled through the prickle-cell layer making tunnels through it. (Civatte) When they reach the barrier of the horny layer these groups of cells spread out forming lenticular masses resembling small, dry abscesses. In the smaller of these the cells are mononuclear in the larger polymorphonuclear. In occasional cases on the palms and soles, and very rarely elsewhere these abscesses are so pronounced as to be visible to the naked eye as flat yellow "lakes" of pus situated in the thickness of the epidermis. They are always sterile on culture.

**SIGNS AND SYMPTOMS** The lesions of psoriasis vary in size from minute papules only just visible to sheets covering large areas of the body. Often they are discs, from a half to several inches across which may be more or less circular or else irregular in outline. Sometimes these heal in the centre leaving rings which extend into gyrate figures.

The lesions are reddish and slightly raised they are covered with more or less scale which becomes silvery on scraping. If the scraping is continued the scales all come off leaving a smooth



FIG. 154

Psoriasis. Arthropathic Type Forearm. N to swelling of wrist.



FIG. 153.

Psoriasis. Arthropathic Type. Note periarticular swelling and pitting of nails.

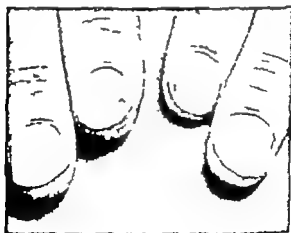


FIG. 156.

Psoriasis of Nails. Pitted Type

red surface on which a number of small bleeding points soon appear. These are the tops of the capillary loops exposed owing to the thinness of the prickle-cell layer over them. The lesions of psoriasis are always dry and very rarely become infected. They seldom irritate much.

Psoriasis generally attacks the extensor surfaces in preference to the flexor but there is a type which is confined to the flexures (groins, perinaeum axillae, toes) and appears as smooth red areas without scales, but with the usual sharp edges. This type often itches considerably. The scalp is often affected, and



FIG. 157

Psoriasis of Nails. Note separation of distal portions from nail beds.

the eruption tends to spread on to the forehead. The rest of the face, however, is comparatively seldom attacked. The nails may be affected in three ways, either by showing rows of small pits like those on a thumb, or by the nail becoming detached from its bed in the distal half or three-quarters of its length or by its becoming rough, thick, yellow and friable. The palms and soles may be affected, usually symmetrically. The thickening and scaling may be very great and may cause deep and painful fissures owing to the loss of elasticity of the thickened horny layer. On the palms and soles the eruption may be pustular constituting the so-called *acrodermatitis psoriatica* (Fig. 161 and p. 337) and in these cases there may or may not be psoriasis of the ordinary type elsewhere.

Most cases of psoriasis are very chronic but fairly often one sees an acute attack in which an eruption of very small spots



comes out rapidly all over the trunk (*guttate psoriasis*). In such cases there is frequently a history of an acute tonsillitis immediately preceding the onset of the psoriasis.

*Arthropathic psoriasis* is a form of the disease in which it is



FIG. 155.

Psoriasis of Nails. Thick, rough, friable type.

associated with a type of erosive arthritis very similar to rheumatoid arthritis, except that the distal interphalangeal joints are affected in over half the cases. The nails are also involved in over 80% and it is with this, rather than with the cutaneous lesions that the arthritis varies the two remitting and relapsing together.

**DIAGNOSIS** The diagnosis is usually easy but occasionally difficult. The points to look out for are the dryness, silvery scaling and sharp edges characteristic of psoriasis. Scraping the lesions until the typical smooth red surface with bleeding points appears is sometimes of assistance.

Psoriasis when not typical may have to be diagnosed from *seborrhoea* (dandruff) of the scalp *seborrhoeic dermatitis* elsewhere, *sypilis* both papular secondary and circinate tertiary

types, *psoriasis rosacea*, *lines circinata* lichen planus eczema on palms and elsewhere, ringworm, eczema and syphilis of nails,



FIG. 158

Psoriasis of Fingers and Palms.

and the flexural types of psoriasis from *lines cruris intertrigo* and *arborescent dermatitis*. From *dandruff* (p. 368) the principal points are that the edges of psoriasis patches are sharp whereas those of dandruff patches are indefinite. Either disease may involve the whole scalp, but the characteristic edges will still be

seen at the hairy margins of the scalp. The scales in psoriasis are drier and more silvery than those of dandruff which tend



FIG. 167

PSORIASIS OF SOLES. Ordinary Type

to be moister and yellower. The same points hold good in the distinction of psoriasis from *seborrhoeic dermatitis* (p. 353) else-



FIG. 161

*Pustular Psoriasis of Sole.*      *Acrodermatitis Perstans.*

where on the body with the addition that in seborrhoeic dermatitis there are usually beyond the edges of the patch, outlying, small, red, follicular papules. The distribution of the lesions

also tends to be different, seborrhoea usually affecting the flexures, or the middle line of the chest and back while psoriasis is more common on the extensor surfaces.

A *scaly papular secondary syphilide* (p 201) is sometimes difficult to distinguish from psoriasis. The principal points in favour of syphilis are the presence of other signs of syphilis, adenitis, mucous patches, anaemia, positive Wassermann reaction, etc. the presence of lesions on the central parts of the face a flexor as opposed to an extensor distribution of the lesions, and the results of scraping the papules. When the scales have been scraped off a psoriasis patch very little infiltration can be felt, in a syphilitic papule there is still considerable infiltration. A syphilide does not yield the smooth red surface with bleeding points characteristic of psoriasis but a yellower surface, small parts of which tend to come away on the blade of the scraper. A *scaly circinate tertiary syphilide* (p 201) also may have to be distinguished from psoriasis and this may be very difficult. In the tertiary syphilide some infiltrated nodules can generally be found, especially at the periphery and there may be a small crust here and there. There will usually be some signs of scarring on the healed areas in the centre, whereas psoriasis, though it may cause temporary pigmentation or depigmentation, never causes scarring. Assistance may be obtained by finding definitely recognisable syphilitic lesions or their scars elsewhere e.g. chronic glossitis. The Wassermann reaction is not reliable here because 20% of tertiary syphilitics give a negative result. It may be necessary to try the effect of antisyphilitic treatment in order to arrive at a diagnosis.

*Pityriasis rosea* (p 330) may be very difficult to diagnose from certain types of generalised small pattern psoriasis. In pityriasis rosea a history of a herald patch may be obtained and some of the lesions, probably those on the flanks, will show the characteristic centrifugal scaling. Psoriasis tends to occur on the distal portions of the limbs to a greater extent than does pityriasis rosea. It is extremely rare for a patient to get a second attack of pityriasis rosea whereas psoriasis commonly recurs.

*Tinea circinata* (p. 150) may occasionally resemble a circinate psoriasis but it has a much greater tendency to show pustules or vesicles, and the fungus can generally be found microscopically

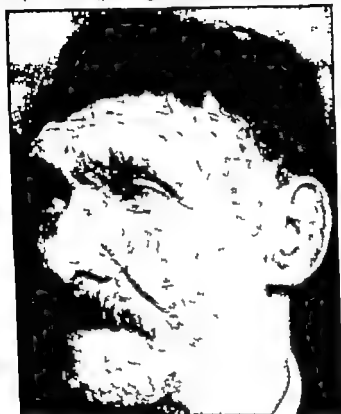


FIG. 162

Psoriasis of Face. S.E.—Face, part from forehead to chin, is comparatively seldom affected.

*Lichen planus* (p. 361) is not infrequently confused with psoriasis, but if a careful examination be made in good day light the violaceous colour of lichen planus as opposed to the red brown colour of psoriasis will be a help. In lichen planus it is nearly always possible to find somewhere the characteristic polygonal, flat topped shiny papules, e.g. on the front of the forearms or wrists. Small white spots or streaks of lichen

planna may be found inside the cheeks in some cases, or papules or small annular lesions on the penis. Lichen planus also tends to itch more than psoriasis.

Psoriasis of the palms and soles has to be distinguished from eczema syphilis, and ringworm. Eczema (p 322) like psoriasis, is usually bilateral the type of *tertiary syphilis* (p 264) likely to cause confusion is generally unilateral.

It may be very difficult to distinguish between psoriasis and chronic scaly eczema of the palms, in the absence of recognisable lesions elsewhere on the body and these are not necessarily present in either disease. In eczema some evidence of vesicles may be discoverable at the margins of the scaly patches.

Ringworm (p 166) can only be diagnosed with certainty by finding fungus. It is much less frequently a cause of scaly lesions on the palms and soles than is psoriasis.

Lesions which may be confused with flexural psoriasis are *tinea cruris intertrigo* and *seborrhoeic dermatitis*. *Tinea cruris* (p 162) must be diagnosed by finding fungus macroscopically. *Intertrigo* (p 184) has not got the uniform smooth red surface or the sharp margins of flexural psoriasis, and the same applies to *seborrhoeic dermatitis* (p 390). In flexural psoriasis ordinary lesions of the disease may or may not be present elsewhere.

PROGNOSIS. Any given attack can usually be cured but it is impossible to say how long an interval of freedom is likely before the next attack. It may be weeks months, or years. If any lesions are left, e.g. on the scalp the interval is likely to be shorter than if every trace of the disease were removed.

TREATMENT. Diet has little or no effect in the treatment of most cases of psoriasis except that alcohol tends to aggravate the disease. I have however seen two cases in which a low fat diet did seem to be followed by improvement in the psoriasis. Acute cases should be kept in bed and treated with bland local applications such as zinc cream or paste or a simple paraffin ointment. When the disease has ceased to erupt hydrarg ammon gr x. (20%) and Liq picis carb 3 i (100%) may be added to each ounce of the ung paraffini. This should be thoroughly rubbed in night and morning.

In an acute case, or one in which fresh lesions are still appearing, irritating applications such as dithranol and tars should be avoided because they may only increase the eruption.

In a chronic case if extensive, the patient should be kept in bed. The scales may be removed daily by washing with soap and water in a bath to which 2 to 4 ounces of a solution of coal tar has been added. The affected areas are treated twice daily with  $\frac{1}{2}\%$  dithranol in Lassar's paste, the strength of the dithranol being slowly increased to 1 or even 2% on the stubborn patches. A lower strength should be used on flexural lesions and it is safest to begin with 0.1% in equal parts of zinc paste and soft paraffin. This treatment is greatly augmented by daily first degree doses of ultra violet light. In certain individuals, dithranol causes an acute oedematous erythema, which is the reason for beginning with relatively weak strengths. It also causes a greyish discolouration of the surrounding skin (but not of the psoriatic lesion). This soon disappears on cessation of the treatment, but the violet staining which it causes on linen is permanent. Finally if allowed to get into the eye, it causes a severe conjunctivitis. Notwithstanding these disadvantages, dithranol is the most effective local application in the treatment of psoriasis. If adequate provisions are made in hospitals, and patients are supplied with mullin undergarments to prevent staining of their clothing, treatment with dithranol and ultra violet light can be carried out on out patients, either daily or less frequently.

The second most effective application is tar used as prepared coal tar in a strength of 2 to 6% in a paste, e.g. White's tar paste (p. 333). In certain mild cases a weaker tar application may be used as ung. Hyd. Ammon. Co.

Hydrag ammon.	gr x.	2
Liq picis carb	3 i.	1℥
Ung. paraffini B.P. ad	3 i.	100

The following ointment is also helpful, especially in psoriasis of the palms and soles.



R	Ol. cadini	3i.	12
	Ac. salicyl.	gr xxx.	6
	Ung. paraffini B.P.	3i.	100

Very severe and extensive cases of psoriasis are sometimes improved by protein shock treatment (p. 24) T.A.B. vaccine (50-800 millions or more) being injected intravenously once a week. When the condition evolves into one of erythrodermia systemic steroid hormone therapy is necessary. This frequently causes an initial improvement but as the skin clears classical lesions of psoriasis reappear which must then be treated along routine lines.

In psoriasis of the scalp the head should be washed two or three times a week with spirit soap, or a soapless shampoo, and the following ointment rubbed in daily through partings.

R	Hydrarg. ammon	gr x.	°
	Ac. salicyl.	gr xx	4
	Ol. cadini	3i.	1°
	Benzoinated Lard or		
	Haklen's Emulsifying Base	to 3i.	100
	or		
	Haklen's Emulsifying Base	5i.	25
	Water to	3i.	100

Liquor pruri carbonis may be substituted for the ol. cadini but is less effective. Dithranol may also be incorporated in a strength of  $\frac{1}{4}$  to  $\frac{1}{2}\%$  but should only be used on dark haired individuals since fair hair is stained by it. Small doses of X rays (100r) are of use here in helping to remove chronic patches.

X RAYS and especially Grenz rays, are of the greatest value in the treatment of localized patches of psoriasis especially in assisting an ointment to remove a chronic patch and so encouraging the patient. In a chronic recurrent disease like psoriasis care must be taken not to use too much X rays on any given area especially as the skin of psoriatics is more liable than normal skin to develop telangiectases. In psoriasis of the palms and soles X rays are invaluable except in the pustular type (acrodermatitis perstans) in which they seem to have little effect.

They are the only treatment of any use for psoriasis of the nails.

ULTRA VIOLET LIGHT is extremely effective in psoriasis. It is given in fair degree erythema doses daily if possible combined with local applications of dithranol or tar.

### PITYRIASIS ROSEA

A common disease characterized by an eruption of pink oval macules on the trunk and upper parts of the limbs which tend to scale from the center outward.

ERUPTIVE CAUSE. It is probably an infectious process.



FIG. 102.

Pityriasis rosea. Rose.

by a filter passing virus, for second

On the other hand the patient is except in the severest cases when sore throat and adenitis. It is rare to case transmission, and inoculation

seldom been successful. Most of the patients are young (6-30 years) the sexes are equally affected, and the maximum incidence is in the last three months of the year.

**PATHOLOGY** Dilatation of vessels with oedema and round-cell infiltration in the papillae and the sub-papillary layer of the corium. Oedema of the epidermis leading to the formation



FIG. 164

*Pityriasis rosea*. Note long areas of macules parallel with the ribs.

of microscopic vesicles containing mononuclear leucocytes. Parakeratosis.

**SIGNS AND SYMPTOMS.** The first lesion is usually the "herald patch," a red scaly macule up to  $1\frac{1}{2}$  inches in diameter situated on some part of the trunk or upper parts of the limbs. When this has been present about a week the general eruption appears suddenly all over the trunk and the upper parts of the limbs. It may exceptionally extend up the neck, on to the scalp and

PLATE VI



*Pityrasia rosea*



sides of the face, and also on to the forearms and legs. The general eruption consists of two types of lesions, small red follicular papules and the pear oval macules characteristic of the disease. These are a  $\frac{1}{2}$  of an inch in length, oval, and on the thorax tend to have their long axis parallel to the under



FIG. 143.

Pityriasis rosea. Macular and follicular lesions.

lying ribs. After these pink macules have been present for a few days they tend to become buff-coloured and rather wrinkly in the centre and then begin to peel from the centre outwards. Typically a ring of scales is formed whose free edges are towards the centre and attached edges towards the periphery of the lesion. Not all the lesions will show these characters, but if the flanks are examined probably some fairly typical spots will be found.

The macules may exceptionally be of much larger size and in rare cases vesicular. In some cases typical macules may be almost absent the red follicular papular part of the eruption being very prominent. Itching is very variable but is usually slight or absent. The disease is self limited, usually lasting six to eight weeks, and second attacks are very rare.

**DIAGNOSIS.** The eruptions most liable to be confused with pityriasis rosea are a small pattern *psoriasis* a *macular secondary syphilide* and a scattered *scabiorrhoea corporis*. The herald patch may be mistaken for *linea curvata*. In *psoriasis* (p. 342) the lesions do not show the centrifugal scaling characteristic of pityriasis rosea, and the scales tend to be attached centrally and free peripherally the converse of what happens in pityriasis rosea. There is, however a type of *psoriasis* papule which is surrounded by a ring of inwardly directed scales. In *psoriasis* the scales become more silvery on scraping and if all the scales are scraped off the characteristic smooth red surface with bleeding points is left. *Psoriasis* is more likely to occur on the distal portions of the limbs than is pityriasis rosea. A history of previous attack would be much in favour of *psoriasis*.

The *macular secondary syphilide* (p. 253) is often confused with pityriasis rosea. In the syphilide there is no herald patch the primary sore may be discoverable the macules are as a rule rounder and more uniform in size than those of pityriasis rosea they may be rather infiltrated and are less likely to be scaly. They are more likely to be present on the flexor aspects of the forearms and on the palms and soles. Lesions on the palms and soles very rarely occur in pityriasis rosea. With a secondary syphilide other signs of the disease can generally be

discovered, if looked for. These may be nocturnal headaches, anaemia, "moth-eaten" alopecia, mucous patches in the mouth, injected fauces, laryngitis, adenitis in posterior triangles



FIG. 109  
Pityriasis rosea. Follicular Type.

of the neck at the elbows and in the groins and possibly a primary sore or condylomata. The Wassermann reaction is not necessarily a help because pityriasis rosea is fairly often seen in patients who are under treatment for syphilis and may



therefore have a positive Wassermann reaction in any case. I can recall at least two patients who developed pityriasis rosea during their treatment for secondary syphilis and were naturally very depressed at seeing a fresh eruption of what they supposed to be syphilis.

A *scattered seborrhoea corporis* (p 161) is most likely to be distributed chiefly over the sternum and between the scapulae, and the patches will be less regular in shape than those of pityriasis rosea. The face and scalp are more likely to be affected in seborrhoea corporis than in pityriasis rosea and a history of previous attacks would be strongly in favour of seborrhoea corporis.

The herald patch of pityriasis rosea is often mistaken for a patch of *tinea circinata* (p 390). The latter however is generally more inflammatory its outline is sharper and its colour redder. With a lens the edges of the tinea patch can generally be seen to consist of tiny vesicles or pustules. The herald patch is always on a covered part of the body while tinea need not be. A microscopic examination will probably reveal fungus in scales from the tinea but never in those from the herald patch.

**PROGNOSIS** The disease gets well without treatment in six to eight weeks and second attacks are very rare.

**TREATMENT** In the majority of cases the disappearance of the disease can be hastened by ultra violet light baths of sufficient intensity to cause peeling along with the use of ung. acid. salicyl. 2% at night. If the light is not available, soaking for twenty minutes every night in a bath the water of which is coloured dark pink with potassium permanganate and the subsequent application of ung. acid. salicyl. is also effective. The staining of the bath by the potassium permanganate may be removed with a solution of sodium thiosulphate or with a cream made by mixing potassium bitartrate (cream of tartar) with hydrogen peroxide solution (R. J. Morgan and R. M. Balcreat *Arch. Derm. & Syph.* 1931 69 101). All that is needed however in the great majority of cases is a simple antipruritic such as 2% phenol in calamine lotion.

## CHAPTER XVII

### LICHEN PLANUS

A disease characterised by an eruption of small papules which are polygonal flat-topped shiny and usually of a bluish pink colour.

**ETIOLOGY** Unknown, although Kyrle believed it to be due to a filter passing virus affecting primarily the epidermis, of warts, molluscum contagiosum, psoriasis. These conditions as well as lichen planus are liable to occur along the line of a scratch. Lichen planus often appears to have some relation to prolonged nerve strain, worry or insomnia. It generally occurs between the ages of thirty and sixty women being affected more often than men in the ratio of three to two.

**PATHOLOGY** It is uncertain whether the first change takes place in the epidermis or in the corium. Kyrle thought the first change perceptible was a multiplication and degeneration of the prickle-cells in the epidermis. the majority of observers however think that an inflammatory dilatation of the vessels in the corium is visible earlier. When fully developed the changes in lichen planus papules are a well-defined edge to the lesion hyperkeratosis without parakeratosis (p. 5) stratum granulosum thicker than normal in some places and absent in others increase in thickness of the prickle-cell layer but without exaggeration of the interpapillary processes, which are ill-developed indefinite outline between epidermis and corium vesicles here and there between epidermis and corium dense small round-cell infiltration in the papillary layer of the corium very sharply limited below dilatation of vessels in the papillae and the subpapillary layer of the corium destruction of the elastic tissue and degeneration of the collagen.

In old lesions the centre of the papule becomes depressed by the atrophy of the epidermis and the flattening out of the

papillae so that eventually in some cases, a thin, atrophic epidermis rests directly on the massively infiltrated upper layer of the corium in which the elastic tissue and collagen have

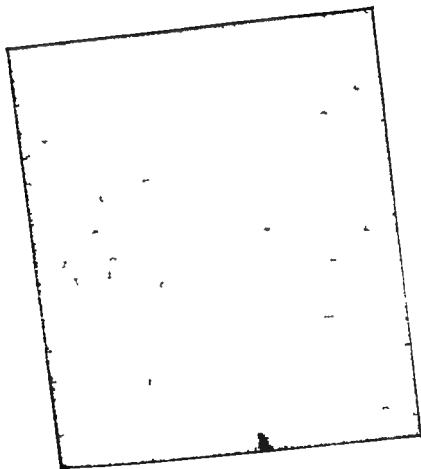


FIG 167

Lichen planus showing well the characteristic flat topped, polygonal, shiny papules

largely degenerated. At this stage there is a considerable amount of melanin in the basal cells of the epidermis and in chromatophores in the corium causing the brown colour of

PLATE VII



Lachen Plains





*Dr. Adenon, Oms.*

FIG. 162.

Lichen planus. Typical lesions on forearm.

old lichen planus lesions. In the lichen plano-pilaris (lichen acuminatus) type of the disease there is great hypertrophy of the horny layer at the mouths of the hair follicles forming pointed, horny spines. In the lichen verrucosus type there are horny plugs embedded in the general thickening of the horny layer and marking the mouths of the follicles and in some cases of the sweat ducts.

**SIGNS AND SYMPTOMS.** Sub-acute or Chronic Localized Type The usual case of lichen planus exhibits a number of small flat-topped, polygonal, shiny papules a millimetre or two in diameter and of a bluish pink, violet colour on the anterior surface of the fore-arms the front of the legs and the inner sides of the thighs.

Some of the papules are discrete while others have coalesced to form slightly scaly plaques. Many of the individual papules have rather depressed centres while some of the larger ones show a mottled appearance owing to alternate areas being white

and violet respectively. These areas are known as the *strias of Wickham*. The white areas are those where the granular layer is abnormally thick the violet ones where it is absent and so allows the capillaries beneath to show through. The eruption may itch severely or hardly at all.

In about half the cases of lichen planus dead white spots and streaks are seen on the inner surfaces of the cheeks. These cause no symptoms. Bald areas may occur on the tongue leading to a mistaken diagnosis of leukoplakia. The nails are involved in about 10% of cases, the appearance varying from a diffuse



FIG 169

Lichen Planus. Annular form on palm.

pitting or vertical ridging, to progressive thinning and eventual complete loss of the nail plate. The lesions of lichen planus have occasionally been seen in the nose, larynx, urethra, colon, rectum and anus. Also in the bladder on the gastric mucosa



FIG. 170.

*Lichen verrucosus. Front of Leg.*



and on the tympanic membranes (Warin et al *Brit Jour Dermat & Syph.*, 1948 60 249)

The eruption of lichen planus may be either much more extensive than described above, or it may be more restricted. Sites commonly affected are, in men the penis, in women the vulva and anus and about the waist. The disease begins insidiously and progresses slowly for months and sometimes years. When retrogression begins the papules take on a browner colour which may make them more noticeable to the patient but is a hopeful sign. The pigmentation may remain for months after the papules have disappeared.

**VARIATIONS.** *Annular form* *Lichen annularis* This is not uncommon. Some of the papules instead of forming plaques form rings  $\frac{1}{2}$  to  $\frac{3}{4}$  inch in diameter. These rings are usually made up by the coalescing of individual papules. Less commonly the ring is formed by the extension outwards of a single papule which heals in the centre while extending at the margins.

*Verrucous form*—*Lichen verrucosus* *lichen hypertrophicus* This is commonly seen on the legs but may occur elsewhere. The lesions are thick raised plaques from a half to several inches across and covered with horny scales. Often there are minute horny plugs at the orifices of the sweat ducts and hair follicles and when these fall out they give the surface a pitted appearance. The colour is usually greyish violet. They itch a great deal and the consequent rubbing no doubt tends to perpetuate them for they are extremely resistant to treatment.

*Spiny form*—*Lichen plano-pilaris* In this form some of the lesions occur at the hair follicles and form prominent or short horny spines. A group of these feels like a nutmeg-grater. The neck is the usual site for this form of eruption.

*Linear form* In this rather uncommon form the lesions, which are of the usual type, occur in a long line or lines possibly down the whole length of a limb. In some such cases the line appears to correspond with the distribution of a nerve in others it does not. This type has to be diagnosed from a segmental warty naevus. The history will usually assist as a naevus is

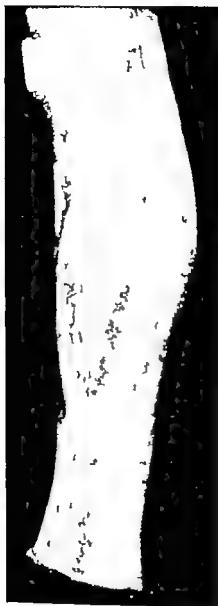


FIG. 171  
*Lichen planus. Linear form.*

likely to have been present from birth or soon afterwards. When lichen planus occurs in children it is comparatively frequently of the linear type.



FIG. 172.

Lichen planus. Generalised Type.

**Atrophic form—lichen planus atrophicus** In this form marked atrophic changes occur and groups of small depressed white scars are left. It may be difficult to distinguish these from the lesions of the punctate type of scleroderma—white spot disease and from those of lichen sclerosus vel atrophicus (p. 179)

Lichen planus seldom occurs on the scalp but Graham Little and Barber believe that some cases of cicatricial alopecia are really atrophic lichen planus occurring on the scalp (p. 374)

Vesicular form This is occasionally seen, especially after the administration of arsenic. The bullae may be from  $\frac{1}{2}$  to 1 inch in diameter. They are due to an exaggeration of the microscopic vesicles which normally occur in the disease (vide Pathology p. 361)

Acute generalised type This type is less common than the chronic localised form. It may take a week or two to appear but sometimes does so with great rapidity practically the whole body being involved in less than twenty four hours. The lesions are very numerous and may be closely set over large areas of the trunk and limbs. They are at first redder than the papules of the chronic form but later take on the usual violet tint. The irritation may be severe and prevent the patient from sleeping, and if this continues for some weeks mental symptoms may supervene. Fortunately this type of the disease usually runs a shorter course than the chronic localised type and generally subsides rapidly if the patient is put to bed and kept there. Relapses and recurrences may however occur

DIAGNOSIS. *Chronic lichen planus* from *psoriasis* (p. 342) In psoriasis the colour is a brighter red and the scales more silvery especially on scraping. When the scales are scraped off a smooth red surface is left with numerous small bleeding points. The papules have not got the polygonal shape nor the flat top with depressed centre characteristic of lichen planus nor do they show the striae of Wickham.

*From papular eczema* (p. 322) In eczema the papules are rounded, oedematous looking, and may show a tendency to turn into vesicles. In lichen planus they are polygonal flat topped and essentially dry and hard looking.

*From lichenification* (p. 237) This may be very difficult even with the assistance of a section. It usually has to depend upon whether undoubted lesions of lichen planus can or cannot

be found elsewhere, e.g. mouth or penis. The principal histological differences are that in lichenification there is parakeratosis (persistence of nuclei in horn cells) as well as hyperkeratosis, whereas in lichen planus there is little or no parakeratosis. The papillae and interpapillary processes



FIG. 173.  
Lichen planus, tongue.

are exaggerated in lichenification, instead of being less marked than normal as in lichen planus. In lichenification the cell infiltrate in the corium is not so sharply limited deeply as it is in lichen planus.

*Verrucose lichen planus* from lichenification (vide above). To some extent the situation is a help lichenification being particularly common on certain sites, e.g. back of the neck flexures of elbows, and knees. Lichen planus verrucosus is more likely to be found on the shins or about the ankles and is also more likely to show a number of small horny plugs on the surface.

*From lupus verrucosus* (p. 207). The lesions of lupus are monster more papillomatous and probably crusted in places. In lichen verrucosus the lesions are essentially dry and scaly.

*Linear lichen planus* from segmental scaly naevus (p. 67). The naevus is of slow development and has probably been present since birth or soon after. Lichen planus comes out relatively

rapidly later in life. Typical lichen planus papules may be found elsewhere.

*Mucous membrane lesions of lichen planus from syphilitic mucous patches* (p 257) These are bluish, oval, and tend to be superficially ulcerated. Lichen planus lesions are dead white in colour and form small spots and fine streaks. With mucous patches other lesions of syphilis will probably be found if looked for.

*From leukoplakia* (p 264) This forms much larger sheets and the lesions are thicker. They occur further forward in the mouth, near the angle of the lips. Lichen planus lesions inside the cheeks are more likely to be opposite the back teeth.

*Acute generalised lichen planus* may on occasion have to be diagnosed from one of the *erythrodermias* (p 452) an *extensive dermatitis* or *eczema* or even from an *acute specific fever*. In all such cases a careful examination in a good light will reveal the papules with their characteristic shiny flat tops.

*From pityriasis rubra pilaris* (p 469) Here the papules are of a reddish brown colour circular not polygonal round or conical not flat-topped, there is much more scaling, and the backs of the proximal phalanges of the fingers generally exhibit the typical conical papules of pityriasis rubra pilaris.

*Pachoxosis* About a quarter of the sub-acute or chronic cases clear up in 6 months and two thirds in a year but some may last for 3 or 4 years. Recurrences occur in about a fifth. The acute cases run a shorter course of four to six weeks if efficiently treated but here again relapses and recurrences may take place.

**TREATMENT** In an acute case the patient should if possible be sent to bed. Systemic steroid therapy has a dramatic effect, clearing the eruption in three to four weeks, and rarely is a daily dose of more than 30 mg of prednisolone necessary. Unfortunately however this treatment is only suppressive and maintenance doses have to be continued for as long as the disease is active.

In a chronic case a holiday is one of the most useful treatments. Widespread or resistant cases may again have to be treated with steroids. It is doubtful if any other form of therapy has a direct effect on the disease but those which have been

advocated are Liq hydrarg perchlor 5 i. (4.0 m.l.) Aqua ad 5 ½ (15 m.l.) t.d.s p.o. ex aqua Vitamin B1 and Enecol" (mercury salicyl arsenate) 2 m.l. containing 0.06 gm., given deep into the muscle two or three times a week for 11 weeks. Gentle sedation with amylbarbitone gr ½ two or three times a day may also help.

**LOCAL TREATMENT** Local treatment in chronic cases depends on whether there is much itching or not. If there is, simple antipruritics (p. 24) such as 2% phenol in calamine lotion or Eurax ointment may be used. X rays are generally very useful if applied to chronic patches, but I have several times seen their application followed by a fresh eruption of lichen planus papules covering the area X rayed when the case was too recent.

Verrucose patches such as occur on the legs are often extremely resistant to treatment. X rays here are generally useful but often fail, even after two or three full doses. They are always worth trying however. The horn thickening may be reduced also by the use of salicylic acid plasters or ointments (up to 50%) or by rubbing with Liq potassae. I have had good results in two cases by painting the verrucose lesions with Thorium X (1200 c.a. units in 1 c.c. of alcohol) every two or three weeks and after painting varnishing the lesions with collodion (v p. 47). Sometimes hydrocortisone (25 mg per m.l.) injected into the lesion is effective. Half a m.l. mixed with an equal quantity of hyaluronidase is injected fortnightly.

I have never myself seen any benefit from lumbar puncture or from X ray treatment of the spine in lichen planus.

## CHAPTER XVIII

### DISEASES OF THE HAIR AND NAILS

The hair may be congenitally absent in cases of congenital ectodermal defect or thin and scanty in ichthyosis follicularis.

Loss of hair (alopecia) is a common condition and may be confined to localized areas of the scalp or beard, or may extend over the whole scalp or even the whole body. The causes of loss of hair may be classified as follows

Diffuse loss on the frontal regions and vertex.

Senile alopecia (p. 374).

Hereditary premature alopecia (p. 374).

Seborrhoea oleosa (p. 337).

Pityriasis capitis (dandruff) (p. 338).

General thinning or loss of hair on scalp.

Debilitating diseases, e.g. anaemia, diabetes, phthisis.

Following acute febrile illness, e.g. influenza, perturbation.

Thyroid or pituitary defect.

Pityriasis capitis (dandruff) (p. 338)

Alopecia totalis (p. 376).

In some general skin diseases e.g. erythrodermia (p. 455)

After ingestion of thallium acetate

After X-ray treatment (p. 177)

"Moth-eaten" appearance on back and sides of scalp.

Scyphus (p. 36).

Loss of hair in well-defined patches on the scalp

Alopecia areata (p. 376).

Ringworm (p. 170).

Favus (p. 180).

Staphylococcal infection (p. 197).

Streptococcal infection (erysipelas) (p. 190).



Cicatricial alopecia ("pseudo-pelade") (p. 374).

Due to rubbing or pulling of the hair (trichotillomania) (p. 383)

#### Scars.

Favus (p. 180)

Kerion (p. 172).

Lupus erythematosus (p. 308).

Scleroderma (p. 435)

Herpes zoster (p. 226).

Syphilis (p. 261)

Lupus vulgaris (p. 205)

Boils (p. 187)

Following excessive X ray and radium treatment (p. 80)

Injury

Burns and scalds.

#### Complete loss of hair on scalp and body

Alopecia universalis (p. 310)

Most of these conditions are dealt with in other parts of this book or in text books of medicine or surgery. Here we need only consider senile alopecia, hereditary premature alopecia, cicatricial alopecia (pseudo-pelade) and alopecia areata with its varieties alopecia totalis and alopecia universalis.

Senile alopecia occurs at very various ages in different individuals and is accompanied by whitening of the hair. It is much more common in men than in women, and affects the vertex and frontal regions first and most. The affected scalp is smooth and shiny. No treatment has any effect upon it.

Hereditary premature alopecia occurs on the vertex and frontal regions soon after the age of twenty years in the males of certain families. It is often difficult to distinguish from alopecia due to dandruff (p. 388) or to seborrhoea oleosa (p. 387) which affects the same areas. It is generally worth treating such a case as one of dandruff even if no scaling can be seen, because quite often treatment with sulphur and salicylic acid will arrest the fall of the hair showing that the condition was due to seborrhoea and was not really hereditary.

Cicatricial alopecia. Folliculitis decalvans ("pseudo-pelade"). It is probable that several different conditions are grouped under

this title. One form occurs most commonly in adult males and another in middle-aged women. The disease usually attacks the upper part of the scalp. It is of insidious onset and slow evolu-



FIG. 174

Cicatricial alopecia. "Pseudo-pelade." Note scarring and disappearance of hair follicles, also isolated tufts of few hairs each.

tion. It appears as small bald patches from which the hair follicles have disappeared and on which the scalp is converted into a depressed atrophic scar. The patches are small to begin with and of irregular size and shape but often run together to

form large bald areas leaving a few tufts of hair here and there. At the edges of the patches some of the hair follicles may be inflamed the degree of inflammation varying from pustulation and crusting (*Folliculite épilante*, Quinquand) to a scarcely perceptible rose tint (*Pseudo-pelade Brocq*). Others of these cases appear to be examples of atrophic lichen planus affecting the scalp for lichen plano-pilaris can be found elsewhere or a history of it obtained.

The DIAGNOSIS has to be made from the other forms of alopecia associated with scarring tabulated above. This is not usually difficult.

PROGNOSIS. Unfavourable. The disease tends to be slowly progressive although spontaneous arrest may take place. The hair never returns on the affected areas.

TREATMENT. Treatment has little effect. It usually takes the form of epilation of the hairs in the inflamed follicles at the periphery of the patches andunction of antiseptic ointments, e.g. Ung. hyd. ox. flav. or Hydrarg. biniodidi gr. ii. to petroleum jelly 3 i. (4%) (Crocker). Wise & Sulzberger report benefit from large doses of Vitamin A 200 000 to 500 000 units daily.

If evidence of lichen planus is found the treatment should be that of lichen planus.

*Alopecia areata*. A disease of unknown causation characterised by sudden loss of hair over circumscribed patches on the scalp beard, or elsewhere. *Alopecia totalis* in which the whole scalp becomes bald and *alopecia universalis* where the whole body is affected are only varieties of *alopecia areata*.

ETIOLOGY. It occurs chiefly between the ages of five and twenty five and is uncommon over forty five. The sexes are equally affected. The disease often occurs in different members of one family at different times and may recur several times in the same patient. It is not infectious. The cause is unknown and as in most diseases of unknown causation there are four principal theories, the infective the toxic the endocrine and the nervous. There is no necessity here to go into the points for and against the different theories. For a dis-

common of them vide Roxburgh, *Clinical Journal* 1929 58 p 421 Suffice it to say that at present alopecia areata is generally believed to be due to some disturbance of the sympa-

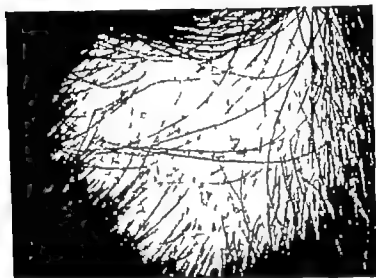


FIG. 17A.

*Alopecia areata. Early active stage. Note exclamation mark hairs.*

thetic system and/or the associated ductless glands. But proof of this is lacking. Many cases are recorded in which alopecia areata has come on from a few days to a week or two after some nervous shock and some, mainly in France after a wound of the face or scalp. In the majority no such association can be discovered. Neither is the mechanism of its production any better understood. Whether the growth of the hair is stopped by some action directly on the cells of the hair matrix, or indirectly via the sympathetic system and the blood supply to the papillae, is not yet settled. Certain French writers maintain that the superficial capillaries are contracted in alopecia areata. My own experiments (*Brit Jour Derm & Syph.* 1929 41 351 and 1931 43 20) lead me to believe that on the contrary the tone of the superficial vessels is less

well maintained in alopecia areata than in the normal skin of the scalp further that even the deeper vessels, from which the hair papillae are supplied are not abnormally contracted, for the skin temperature is no lower on a patch of alopecia areata than on the normal scalp

**PATHOLOGY** Depigmentation of epidermis and lowest parts



FIG. 178.

Alopecia Areata. Advanced stage

of hairs. Epidermis thinned and papillae flattened. Hair follicles empty and atrophied or distorted and dilated to form cysts. About the follicles and small blood vessels is a slight infiltration of round cells and a few mast cells.

**CLINICAL FEATURES** The hair comes out suddenly e.g. over night, or gradually. The patches are completely bald and the skin surface smooth. At the edges of the patches are a few "exclamation mark" stumps. These are hairs broken off about a quarter of an inch from the scalp whose free ends are of the normal colour and thickness but whose inserted ends are very thin and depigmented. The surface of these stumps has the normal shine and their elasticity is almost normal, points which distinguish them from ringworm stumps (p. 173).

The patches may be situated about the top of the head or mainly round the margins of the hairy scalp. They gradually extend at the edges and may coalesce with one another until a large part of the scalp has become bald. The hair when it begins to grow again usually does so first in the centre of the patch i.e. the oldest part of it. The new hair may be of the normal colour or it may at first be white and only regain its normal tint later. In elderly persons, it may remain permanently white. The eyebrows and lashes or beard may also be affected. Sometimes the patches extend until the whole scalp is completely bald (alopecia totalis) or the hair may fall all over the body (alopecia universalis). In severe cases various dystrophic changes may occur in the nails.

**DIAGNOSIS.** From ringworm (p. 170). In this the surface of the affected skin is scaly not smooth. There are large numbers of ringworm stumps all over the patch. In the small-spored type the ringworm stumps themselves have dull surfaces, are often bent in the middle and have lost their elasticity. They fluoresce under Wood's light (p. 44). Ringworm fungus can easily be found microscopically. Small-spored ringworm very rarely occurs in adults, and the large-spored varieties are generally inflammatory. In black-dot ringworm (p. 14) diagnosis depends on finding fungus microscopically.

From syphilitic alopecia (p. 256). In this the patches are

smaller are not completely bald have indefinite margins and no exclamation mark hairs. The general result is a "moth eaten" appearance confined to the back and sides of the scalp. Other signs of syphilis will probably be found if carefully looked for including a positive Wassermann reaction.



FIG 177.

*Alopecia universalis.*

From the various lesions causing scars on the scalp especially cicatricial alopecia. The absence of scarring in alopecia areata the presence of empty hair follicles, and of a few exclamation mark

hairs, and the history will generally lead to a correct diagnosis.

The only other conditions in which exclamation mark hairs occur are both artificial viz. after administration of X-rays or *thallium acetate* for epilation of the scalp e.g. in ringworm.

**PROGNOSIS.** One can never tell when seeing a patient with alopecia areata whether the attack will be of the usual mild variety lasting a few months or whether it will proceed to alopecia totalis or alopecia universalis. The two latter types are much less common fortunately so for the prospects of recovery from them are much less favourable. If the hair shows no sign of re-growth, in an extensive case, after twelve months the prospects become increasingly gloomy but occasionally recovery occurs after years of baldness. Alopecia areata often recurs, later attacks tending to be more extensive and prolonged than the earlier ones.

**TREATMENT.** The cause of alopecia areata being unknown treatment is empirical. Fortunately most cases eventually recover however they are treated. The first thing is to reassure the patient that the disease has nothing to do with syphilis if one suspects that he is worried on this account, as many of them seem to be. The next thing is to get the patient into as good a state of general health both mental and physical, as is possible. A holiday is usually good treatment. If there is co-existing dandruff this should be treated (p. 338). No known method of treatment will check the spread of alopecia areata all that treatment can do is to assist the return of the hair on the already bald patches. Most of the treatments depend for their effect if any on their power of improving the circulation in the bald patch. Painting with liq. epispasticus, carbolic acid, or tincture of iodine or rubbing in lotions containing tr. cantharidini and ammonia or ointments containing chrysarobin are the usual methods. But by far the most effective treatment in my experience is ultra violet light. This should be applied in a big dose once a week to the bald patches, either by means of an ordinary air-cooled mercury vapour lamp or tungsten arc or by the Kromayer lamp applied with pressure to the skin. The dose should be big enough to



cause slight venication or at least pronounced peeling. Regrowth of hair can often be seen to have begun after the second treatment. In extensive cases, or those which resist local ultra violet light treatment, a prolonged course of general ultra violet light baths is sometimes effective. Ultra violet light certainly reddens the skin, i.e. dilates the superficial capillaries and venules more effectively and keeps them dilated for a longer period than any of the other rubefacients mentioned but experiment shows that ultra violet light is not so effective in dilating the deeper arterioles (at the level of the hair papillae) as is an ordinary burn or freezing (Lewis, *Blood Vessels of the Human Skin and their Responses* 1927 p 130) It is known that exposure to ultra violet light stimulates the growth of the epidermis and causes both the prickle-cell and horny layers to become two or three times thicker than normal. (Percival, *Brit. Jour Derm. & Syph.* 1930 42, p 225) As the hair matrix from which the hair is formed is developmentally part of the epidermis, it seems to me natural to suppose that the stimulating effect of ultra violet light on the epidermic cells also affects their near relatives the cells of the hair matrix. The mechanism is presumably a chemical one depending on the breakdown products of the epidermic cells. If these products are carried to the hair papillae by the blood stream this would account for the good effect of general light baths in which the actual bald areas are not exposed to the light. (Roxburgh, *Brit. Jour Derm. & Syph.* 1931 43 pp 20 *et seq*) Dillaha and Rothman found that cortisone by mouth and R A Wilson that ACTH intravenously caused regrowth of hair in a proportion of patients with old-standing alopecia universalis. As growth ceased again on stopping the hormone, this treatment is not of practical use at present.

**Canities.** Whitening of the hair. A white lock of hair is sometimes congenital and inherited. White patches are more usually the result of alopecia areata the new hair not having regained its normal colour. They may also occur in vitiligo. The ordinary senile whitening of the hair generally occurs first on the temples. In some individuals and families this begins

between the ages of twenty and thirty years. It is produced by the disappearance of the pigment from the hair and by the presence of air between the cells of the cortex. The hair is stated to have turned "white in a single night" under stress of great emotion, but such cases, if authentic, are very difficult to explain. It is however recorded that in many cases the hair turned white in five days after the air raids on Coventry in the 1939-45 war.

Canities may develop during an acute illness, or on the area affected by severe neuralgia. White hair rarely regains its normal colour. For information on hair dyes see H. Stanley Redgrove and G. Foan, *Hair Dyes and Hair Dyeing* W. Heinemann, London, 1939.

Trichotillomania is the name given to a habit of neurotic girls and young women of constantly pulling at or rubbing the hair on some portion of the scalp whereby the hairs are broken off short. The bald patches so produced have to be diagnosed from ringworm (p. 170) and from alopecia areata (p. 376).

The points in favour of trichotillomania are that the skin surface is either normal or has prominent follicles and is not scaly as in ringworm and the stumps of the hairs are also normal, not dull, inelastic, and brittle as in ringworm, nor like exclamation marks as in alopecia areata. The patient may also exhibit other artificial dermatoses or may have an anaesthetic palate and sclerotics.

**Hypertrichosis—hirsuties.** Excessive growth of hair on parts usually bearing only lanugo hairs. Congenital hypertrichosis is seen in localised form in hairy moles. Occasionally it is generalised and such cases are usually to be seen in freak shows as "dog-men" or "ape-men."

Acquired hypertrichosis may occur in both sexes, but it is only in women that it causes trouble. It usually comes on about puberty or else after the menopause. The commonest site is on the upper lip. The growth of hair here is a racial and familial trait commonest in dark haired races and individuals. Another common type is that in which strong hairs grow on the sides of the chin and on the upper parts of the cheeks. All degrees are seen from the presence of only a few hairs to a full beard.

Excessive growth of hair on the face is usually associated

with an excess also on the limbs, and sometimes on the chest back and abdomen. On the last situation the pubic hair instead of being limited above by a horizontal line as is usual in women runs up towards the umbilicus.

Hypertrichosis may be part of an endocrine syndrome e.g. in tumours of the suprarenal cortex, basophilic adenoma of the anterior pituitary (Cushing's syndrome) and ovarian arrhenoblastomata. But such cases are rare and in the ordinary case of hypertrichosis the patient is in perfect health.

**TREATMENT** No internal treatment is of the slightest use. Local treatments may be classified into curative and palliative. Of the former electrolysis or diathermy of the hair follicles is the only safe method. X rays should not be used as it is impossible to destroy the hair permanently by this means without damage to the skin, resulting in atrophy, pigmentation, and telangiectases. One palliative treatment is pulling out the hairs either singly with forceps or a number at once by means of wax, applied warm and ripped off quickly when hardened. A suitable wax is beeswax one part, finely powdered resin four parts. The beeswax is melted and the resin dissolved in it when hot. The resulting wax must be softened by heat before application. Other palliative treatments are cutting off the hairs at the skin surface by shaving or by an electric razor breaking them off by rubbing with a smooth piece of pumice stone daily for a few seconds only or corroding them with depilatories such as barium sulphide or strontium sulphide. A usual depilatory is barium sulphide 3*ii* (2*o*) zinc oxide 3*iii* (38) *pulv amyli ad* 3*℥* (100). This is mixed into a paste with water at the time of use spread over the hairy area and left for five minutes. It is then washed off and the hair comes off with it. Zinc cream or zinc paste should now be applied to repair the damage done to the horny layer by the sulphide. Depilatories are too irritating for regular use by most women for the hair and the horny layer of the skin, being both made of keratin suffer almost equally from the depilatory. Modern commercial depilatories are often based on mercapto-glycollic acid or calcium thioglycollate (1 10%).

which are stated to remove hair without apparent irritation of the skin (R. G. Harry *Modern Cosmetology* 2nd Ed. London, 1944)

There is no evidence that shaving or any of the other measures described increases the strength of the hair. On the contrary there is evidence that epilation by pulling out the hairs and by rubbing them off with pumice stone results eventually in a weakening of their growth. The reasons why shaving is popularly supposed to increase hair growth are that each hair has its own "normal" length and if it is shortened



FIG. 178.

"Ladder deformity" of thumb nails. Duration five years.

by any means it grows very rapidly at first but slower and slower later as it approaches its normal length and that as an adolescent becomes older his beard tends to get stronger in any case.

It may be sufficient to bleach the hair by hydrogen peroxide so that it is less noticeable. For this purpose some 10 vol.  $H_2O_2$  should be put in a saucer along with a piece of litmus paper which will turn red. Liq. ammon. fort. should then be added drop by drop until the litmus paper turns blue. The resulting alkaline  $H_2O_2$  should be applied with cotton wool to the hair until it is sufficiently bleached.

**Diseases of the nails.** The most important diseases of the nails are paronychia (p. 155) ringworm (p. 167) psoriasis (p. 345) eczema (p. 324) lichen planus (p. 64) and syphilis, for which the pages indicated should be consulted. A transverse groove across all the finger nails at the same level, "Beau's line" often results from a serious illness. Koilonychia or spoon nails, i.e. nails depressed in the centre and raised at the edges are in some cases congenital and in others due to chronic hypochromic microcytic anaemia: Treatment of the latter with iron does good to the nails. Leukonychia or whiteness of the nails may occur in spots or streaks or may be complete. The nails may become separated from the nail beds as a result of severe chilblains and may split into lamellae at their free margins as a result of the use of nail varnish or its remover.

Brittleness of the nails is due to various causes, among them cuticle remover (KOH or NaOH solutions). Mild degrees of ill health make nails more susceptible to external factors which increase brittleness such as water soap and alkaline cleansers. Iron metabolism is important. Brittleness is often curable by suitable iron medication. (Silver and Chiego 1940 *J Invest Derm* 3 357) T. L. Tyson (*J Invest Derm.*, 1950 14, 323) claims that brittle nails were cured in three months in ten out of twelve patients by their taking 7 gm. of gelatin daily dissolved in water or fruit juice.

The nails may suffer various dystrophies, most of which are little understood and are not particularly amenable to treatment. A common one is illustrated (Fig. 178)

## CHAPTER XIX

### DISEASES OF THE SEBACEOUS AND SWEAT GLANDS DISORDERS OF THE SEBACEOUS GLANDS

The eruptions known as "seborrhoeic" are so called because the scales were supposed by Hebra to represent dried sebum. It is now known that the scales are in all cases composed only of horn cells and that when they appear greasy the moisture is due to serum not sebum (Sabouraud). Although these eruptions are frequently seen in patients who have actual seborrhoea i.e. an excessive flow of sebum, they may equally occur without sebaceous overactivity and it is probable that an abnormal chemical composition of the fat is the important factor.

Seborrhoea oleosa is a condition of hypersecretion of sebum and is the only condition to which the term seborrhoea can be correctly applied. The subjects of seborrhoea oleosa have large sebaceous glands, especially about the sides of the nose and central parts of the face and are very liable to suffer from acne and "seborrhoeic" eruptions. The males are apt to lose their hair early on the temples and vertex, and the females to complain of hair which becomes greasy a day or two after being washed. The condition appears about puberty in a large proportion of people as a temporary event associated with the increased growth of hair at that time. It usually lasts a few years only. As a permanent condition it appears to be an inborn characteristic rather than a disease and treatment is only palliative e.g. frequent use of soap and hot water and sulphur lotions, e.g.

R.	Zinci sulphatis	gr xiii.	4-6
	Potassae sulphuratae	gr xx	4
	Acetone	3 ii.	25
	Aq. roseae ad	℥ 1.	100

X rays to localised areas, e.g. the face, are of the greatest value in reducing the activity of the sebaceous glands.

Dandruff, scurf, pityriasis capitis seborrhoea sicca are names given to the common condition of the scalp in which it is more or less covered with white scales which fall on the shoulders when the hair is brushed. These are simply flakes of the horny layer of the skin produced in excess. Sometimes the scales, instead of being dry and white, are yellow and look greasy. They are not really greasy however as Sabouraud has shown the appearance is due to their being sodden with serum. There is often some redness of the forehead along the hair margin, the "Corona seborrhoeica." Dandruff causes irritation of the scalp, and is important for two reasons, firstly because patients with dandruff are liable to get seborrhoeic dermatitis on the scalp and other parts, and secondly because they are liable to lose their hair early on the temples and vertex.

**DIAGNOSIS** The principal diseases which are liable to be mistaken for dandruff are psoriasis and in children ringworm. In psoriasis there may be either a scaliness of the whole scalp or more commonly of several patches on the scalp. The points of distinction are that psoriasis patches always have a sharp and definite edge whereas patches of dandruff have indefinite margins tending to fade away imperceptibly into normal skin. Where the whole scalp is scaly these features must be looked for at the margin of the hairy scalp. Psoriasis also sometimes forms a red festooned border to the hair margin on the forehead the "Corona psoriatica." If the rest of the body be examined typical lesions of psoriasis (p 315) may be found usually on the extensor surfaces of the limbs, whereas the lesions of seborrhoeic dermatitis if present tend to be in the flexures. The existence of a flexural form of psoriasis (p 315) must not be forgotten.

Ringworm must be diagnosed by the presence of dull broken hairs which fluoresce under Wood's Light (p. 41), confirmed by the finding of fungus by the microscope (p 169)

The papules of secondary syphilis sometimes appear closely set on the forehead forming the so-called "Corona Venerea."

A general examination of the patient will almost certainly reveal other signs of syphilis and the Wassermann reaction will be positive.

**TREATMENT.** The head should be washed thoroughly with spirit soap or a soapless shampoo and, preferably soft water once or twice a week. Every night or every morning the following lotion should be shaken on all over the head and well brushed in. In the case of women the hair must be parted and the lotion shaken on to the partings.

R	Ac. salicyl.	gr xx	4
	(Hydrarg. perchlor	gr $\frac{1}{2}$ )	(0.1)
	Ol. ric.	℥ v	1
	Ol. lavand.	℥ v	1
	Spiritus meth. indust.	ad ℥ i.	100

In the case of men the lotion may be made into a brillantime by increasing the Ol. ric. to ℥ xxx. or more. In the case of women it may be omitted altogether. In the case of women with very fair or white hair the hydrarg. perchlor should be omitted as it has a tendency to darken the hair especially on permanent waving. As the lotion has to be used for an indefinite period and Spiritus meth. indust. requires a fresh prescription for each bottle, bath Eau de Cologne or bay rum may be substituted, but they do not hold the Ol. ric. in solution.

If the lotion is not successful in removing the dandruff after two weeks or so an ointment or emulsion may have to be used such as,

R	Ac. salicyl.	gr. v-xxx.	1-6
	Sulph. precip.	gr v xxx.	1-6
	Ol. Cadini	℥ xxx	6
	Harden's Emulsifying Balm		
	or Ung. Emulsificans R.P.	℥ ii.	25
	Aquum	ad ℥ i.	100

This should be rubbed in every night and the head washed two or three times a week. An ointment is such a nuisance on the scalp that patients soon get tired of using it and the less effective but more convenient lotion will soon have to be substituted if the patient is to continue the treatment. Another method of



applying sulphur to the scalp is by means of a shampoo containing selenium sulphide ("Selsun"). One or two drams are required for each shampoo and the process is repeated weekly or twice weekly to begin with and then at gradually increasing intervals. Some patients remain symptom free indefinitely by using it only once or twice a month. Thorough rinsing of the scalp is necessary after each application. It is essential to continue the treatment for dandruff for months or years. The lotion used two or three times a week will probably keep the condition in check once it has been thoroughly got under control.

**Seborrhoeic dermatitis** is an acute or chronic inflammation of the skin. The *pityrosporon ovale* is found in the more chronic cases, but is a harmless saprophyte. Non-pathogenic staphylococci are also found in the milder cases, but when severe pathogenic staphylococci and streptococci are easily recovered from the lesions. The disease occurs in two principal forms,

(1) **Seborrhoea corporis**—flannel rash—circinate patches with red edges and yellow centres with outlying red follicular papules, usually on the sternal and inter scapular regions. Similar but more scaly patches may occur on the central parts of the face and on the upper eyelids and the eyebrows.

(2) **Seborrhoeic dermatitis** or **seborrhoeic eczema**—sheets of red inflamed weeping skin on the scalp, neck, and in the flexures. This is probably a "sensitisation dermatitis" due to the skin having become sensitised to the infecting organism.

**Seborrhoea corporis**—flannel rash—consists of circinate figures from  $\frac{1}{2}$  2 inches in diameter with red edges and buff coloured centres, and, usually outlying red follicular papules. The patches are slightly scaly and are most common on the trunk especially over the sternum and between the scapulae but they may occur elsewhere. They irritate slightly. This form most commonly occurs in those who wear the same flannel undergarment night and day. The *pityrosporon* is easily found in the scales obtained by scraping the lesions.

**DIAGNOSIS** The lesions liable to be mistaken for flannel rash are those of *pityriasis versicolor* (p. 180) *pityriasis rosea* (p. 200) *psoriasis* (p. 342) and *parapsoriasis* (p. 392).

In *pyramis verrucosus* the patches are generally widespread over the upper trunk not confined to the centre line and of a café au lait colour without any redness the characteristic



FIG. 179

Seborrheic Dermatitis. Seborrhoea Corporis Type. Sternal Region.

mycelium and spores are easily found microscopically (p. 180)

In *pyramis roseus* the lesions are generally widespread over the trunk and upper parts of the limbs, the patches are of regular shape and fairly uniform in size, being usually oval, and

on the thorax often arranged with their long axes in line with the underlying ribs. Some of them, especially on the flanks, will show the characteristic centrifugal peeling. There may be a history of one patch preceding the general eruption by about one week. No organisms can be found microscopically (p 355)

*Psoriasis* is generally more widespread than *seborrhoea corporis*, and the edges of the patches are more sharply defined. On scraping, the scales of *psoriasis* become silvery and if the scraping is continued a smooth red surface is reached which bleeds from numerous tiny points. There may be characteristic lesions on the extensor surfaces of the limbs or on the nails (p 345). No organisms are found microscopically

*Parapsoriasis "en plaques"* These are uniform plaques of reddish yellow colour and slightly scaly which occur on the trunk and upper parts of the limbs. Their most characteristic feature is complete resistance to any form of treatment. No organisms are found in scrapings. The condition is rare

**TREATMENT** The treatment of *Seborrhoea corporis* is easy. Salicylic acid and sulphur ointment 2%-3% usually removes the lesions quickly. *Pityriasis capitis*, if present must be treated or the trunk lesions are likely to recur

On the face *seborrhoea* causes scaly patches which may be slightly red or may be yellowish and greasy looking. The scaly patches commonly affect the upper eyelids. The yellowish greasy looking patches are most common at the sides of the nose and on the upper lip. There is generally a good deal of dandruff in such patients.

**DIAGNOSIS** From *scaly impetigo* This generally occurs in children. In it there is seldom much redness, the lesions are more scaly and the scales are usually arranged in rows rather suggesting the ridges and furrows of a ploughed field. Scaly *impetigo* is most common on the cheeks and chin and is generally made worse by east winds and by alkaline soaps. Dandruff is not present except as a coincidence.

From *lupus erythematosus* The characteristic plugging of the openings of the sweat and sebaceous glands with horny

plugs, giving a "stippled" appearance, can usually be made out on some part of the eruption in this disease.

**TREATMENT** Ung hvd. ammon. co (p 333) weak sulphur and salicylic acid ointments (1% 3%) and small doses of X-rays, 50r 100r once a week for two or three doses.

**Seborrhoeic dermatitis**—Seborrhoeic eczema when it affects the scalp causes the latter to become swollen and red, and to weep profusely so that the serum soaks the patient's pillow at night and runs down his face and neck during the day. After a time the serum is poured out less profusely and crusts are formed, matting the hair together. In children and adults this condition sometimes becomes chronic and lasts for months or years, a streptococcal and staphylococcal infection being present. In chronic cases a pustular folliculitis of the scalp often develops and spreads to the eyebrows and lashes. Seborrhoeic dermatitis of the scalp often extends on to the forehead, forming a red fringe along the hair margin, the "*Corona seborrhoeica*."

It is difficult to account for the sudden onset of cases of acute seborrhoeic dermatitis of the scalp but sometimes attacks appear to have followed the use of too hot or too irritating a shampoo by a patient with dandruff.

**DIAGNOSIS.** Extensive impetigo and dermatitis due to the hair dye paraphenylenediamine are two conditions which may be confused with seborrhoeic dermatitis of the scalp. In impetigo the lesions tend to be localised although they may be pretty extensive, pediculosis capitis will very likely be present, and there may be typical lesions of impetigo on the face or a parient blister alongside one of the finger nails. When the crust of an impetigo patch is removed the phlyctenular edge of the original blister may be visible.

**Dermatitis from hair dye** is diagnosed from the bronzy black appearance of the hair and from the history. A patch test with the hair dye will help in diagnosis.

**TREATMENT** The first step is to clip the hair as short as possible over the whole head. Crusts may have to be removed by the application of equal parts of ol. olivae and aqua calcis or

wet gauze compresses of lead lotion. These simple measures may bring the condition under control but antibiotics, either local (e.g. 1% neomycin lotion) or systemic are frequently required. This treatment is continued until the weeping has ceased. Gauze or butter muslin dipped in calamine lotion or in zinc cream with or without 0.2% of pyrogallol should then be



FIG. 180

*Seborrhoea Dermatitis. Seborrhoeic Eczema Type*

substituted. When the acutest stage has passed 50r doses of X rays at weekly intervals are of great assistance in clearing up the remaining inflammation. When only scales remain a beginning must be made with ointments such as Ung. hyd. ammon. co. (p. 385) or an emulsion of Acid salicyl. and sulphur 1% (v.p. 389). The concentration of the salicylic acid and sulphur should

be gradually increased up to 3% or the lotion given above for the treatment of dandruff substituted. In cases of chronic seborrhoeic dermatitis of the scalp brilliant green and/or crystal violet 1% in zinc paste or zinc cream, Eau d'Alibour pyrogallol gr i. in zinc paste or zinc cream 5 i. (i.e. 0.2%), tar ointments



FIG. 181

Seborrhoeic Dermatitis of axilla. Seborrhoeic Eczema Type. Note outlying red, follicular papules.

(p. 333), and many other things will have to be tried. The hydroxyquinoline preparations, such as Steroxin Vioform and Quinolol ointments and the emulsion described on p. 193, are

also useful in this stage. In many cases nothing short of epilation of the entire scalp with X rays as for ringworm (p. 177) is effective. Even so the condition may relapse when the hair grows again and the epilation may have to be repeated once or even twice.

Seborrhoeic dermatitis very commonly affects the back of the ears where it is almost indistinguishable from a chronic impetigo and is often associated with painful cracks at the junction of the auricle with the head especially at the top and bottom of the ear. Rapid improvement follows the use of local antibiotics alone or with the addition of hydrocortisone but this must be followed by one of the applications used for seborrhoeic dermatitis of the scalp. X rays are also helpful.

**Seborrhoeic dermatitis of the flexures.** This commonly affects the axillae, umbilicus, groins perineum under pendulous breasts, also the fronts of the elbows and the backs of the knees. It is characterised by a red, possibly weeping, surface with outlying red follicular papules on the neighbouring healthy skin. It may extend from the above sites until it involves the greater part of the body surface.

**Diagnosis.** Seborrhoeic dermatitis of the flexures has to be diagnosed from intertrigo tinea, dermatitis due to the use of depilatories or anti-sweating lotions in the axillae also from flexural psoriasis and eczema. *Intertrigo* does not usually show the outlying red follicular papules characteristic of seborrhoeic dermatitis it is not likely to be so widespread or so symmetrical. *Monilia* or streptococci may be found microscopically.

*Tinea* usually affects the groins but may involve the axillae also. The bends of the elbows and knees are seldom affected. In tinea the progress of the disease is rapid the edge is vesicular or even pustular and is much more acutely inflamed than the area which has been longer affected in the centre of the patch. The irritation is very pronounced. Mycelium is easily found in the roofs of vesicles, or in tags of horny layer from the growing edges of the lesion. *Dermatitis* from the use of depilatories or "anti-sweating" lotions is most likely to be



FIG. 182

Seborrheic Dermatitis. Combination of Seborrheic corpora and Seborrheic  
Eczema Types.



limited to the axillae and can usually be diagnosed from the history. *Flexural psoriasis* (p 315) is characterised by its uniform smooth red surface and sharp edges with absence of any fungus in microscopic preparations. I have had, however one case of flexural psoriasis with a heavy saprophytic infection with monilia superimposed. There may or may not be psoriasis elsewhere.

*Eczema* (p 318) especially the chronic form persisting from childhood the so-called *Beunier's Prurigo* (p 328) often affects the flexures of the knees and elbows, less commonly the axillae and groins. The history, the presence of symmetrical lesions elsewhere, e.g. face the absence of the characters of any of the other diseases just mentioned and the frequent presence of lichenification will assist the diagnosis.

**TREATMENT** In the acute weeping stage antibiotic lotions or wet gauze compresses of  $\frac{1}{2}\%$  aqueous silver nitrate, lead lotion or 2% aluminum acetate. When the condition is dry and scaly hydrocortisone ointment is frequently effective or the more traditional remedies may be tried such as *pig magenta* 1% aqueous gentian violet, or zinc cream with the addition of either 2% ichthyol, 0.2% pyrogallol, 2% sulphur or 4% liq. picic carb. The hydroxyquinoline preparations are also useful (p. 180). Sometimes a dusting powder is preferred such as *Conspersus Zinc Ox. et Ac. Salicyl. B.P.C.* Weekly doses of 20 or 50 and later 100r of X rays, up to four or five times are extremely useful. The affected area should be cleansed by mopping with olive oil or liquid paraffin. It is essential to treat also the co-existing *psoriasis capitis*.

**GENERAL PROGNOSIS OF "SEBORRHOEIC ERUPTIONS"** Most of these except chronic seborrheic dermatitis of the scalp and elsewhere are fairly easily cured but have a distinct tendency to relapse.

**GENERAL TREATMENT** of patients with 'Seborrheic eruptions'. An active open-air life when this is possible plenty of soap and water (but not on existing lesions) and a diet containing no excess of carbohydrates but plenty of protein fruit and green vegetables, are desirable. Underclothing of cotton

or linen (which can be boiled) worn next the skin, with woollen garments outside these if required for warmth, and frequent changes of underclothing, are also important. Ultra violet light or strong sunlight almost invariably aggravates an existing seborrhoeic dermatitis.

Acne vulgaris is one of the commonest skin diseases, very few



FIG. 181.

Acne vulgaris. Severe case. Note blackheads and pustules.

people of either sex reaching adult life without having had some degree of acne at some stage of their adolescence. It occurs chiefly on the face back and chest and is characterised by the presence of comedones or blackheads small superficial sebaceous cysts, papules, pustules, thick walled indolent abscesses and scars.

## 400 DISEASES OF SEBACEOUS AND SWEAT GLANDS

**ETIOLOGY** It appears that acne is due basically to an excessive production of androgens in relation to oestrogens, for androgens stimulate the epidermis and sebaceous glands causing hyperkeratosis and seborrhoea while oestrogens have a contrary action (Barber 1918)<sup>1</sup> In addition there is probably an effect due to differences in the receptor mechanism: *i. e.* in the sensitivity of the epidermis and sebaceous glands to the circulating hormones in different people and in different areas of the body (Bloch 1931)<sup>2</sup> The effect of the hyperkeratosis of the follicles plus the hypersecretion of sebum is to form comedones in many of the follicles. It is only up to this stage that the sex hormones can be held responsible for acne. Later stages are due to secondary causes. Acne occurs about equally in both sexes starting usually at or soon after puberty and being at its maximum about 17 to 18 years of age. It occasionally occurs in infants. The upper age limit is very variable. The majority of patients of both sexes recover between the ages of 20 and 30 years but acne is still common in men over thirty and not rare after forty. In women it seldom lasts beyond the early thirties. The greasy skin which pre-disposes to acne is often inherited. Factors which aggravate acne are the following: insufficient use of soap and water to remove the obstructing layers of horn cells and wash away the excess of sebum; excess of carbohydrate and fat in the diet especially such fat-containing foods as chocolate (containing cacao butter) in most patients, and cheese, bacon and ham in a few; the use of greasy toilet creams and make-up and of oil when sun bathing; dyspepsia, constipation and lack of sufficient fresh air and exercise. Acne however often occurs in otherwise perfectly healthy young people who take a lot of open air exercise. In women acne is usually worse before each menstrual period. Bromides and iodides aggravate acne seriously as is seen when an acne patient takes a proprietary "blood mixture" containing potassium iodide. Tropical climates have a very bad effect on acne the lesions becoming

Barber H. W. (1914) in *Modern Trends in Dermatology* Eds H. M. B. MacKenna, London p. 108

<sup>2</sup> Bloch B. (1931) *Brit Jour. Derm. & Syph.* 43, 81

indurated and cystic and leading to superficial burrowing abscesses and deep-seated cellulitis due to secondary infection.

**PATHOLOGY** Hypersecretion of sebum and hyperkeratosis of

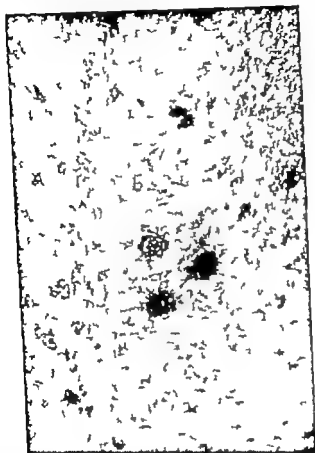


FIG. 184

Acne vulgaris. Back, severe case. Note blackheads, pustules and scars.

mouth of lanugo hair follicle. The result is the comedo or "blackhead" an oat-shaped body composed at its outer end of concentric layers of horny cells and dried sebum with, in the

centre numerous *acne bacilli*, and at its inner end mainly of *sebium*. It often contains a harmless mite the *Demodex folliculorum*. When *staphylococci* are present they tend to be in the superficial layers of the comedo. The outer end of the comedo is dark-coloured due to the presence of a melanin-like

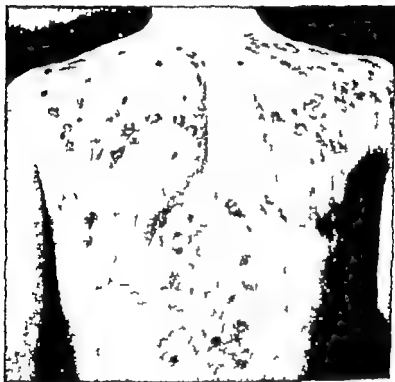


FIG. 183.

*Acne vulgaris* showing severe scarring on back.

pigment. The pressure of the comedo causes some atrophy of the epithelium of the follicle. Sometimes the hyperkeratosis of the mouths of the follicles closes them completely so that small sebaceous cysts are formed rather than blackheads. This cystic type of acne is usually seen on the chin and forehead.

**SIGNS AND SYMPTOMS.** The areas commonly affected by *acne vulgaris* are the forehead, temples, cheeks and chin especially

the sides of the latter the chest and back and also in some cases the deltoid region. In exceptional cases almost the whole body may be covered with blackheads. In such cases there is often intractable suppuration and extensive scarring (*acne conglobata*)

Some blackheads are always to be found, and they may be the only form of lesion present. In some cases they are ex



FIG. 186.

*Acne conglobata. Note extensive scarring.*

tremely numerous, this condition being called *acne punctata*. In most cases some of the lesions progress to red, slightly itchy indurated papules which may either subside slowly or go on to suppuration. When suppuration occurs it may be acute and result in a superficial pustule and a small scar or it may be chronic and result in a small amount of pus enclosed in a very thick walled abscess cavity *acne indurata*. The depressed scars of contiguous acne pustules often give a worm-eaten appearance to the cheeks.

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**DIAGNOSIS** This is usually easy as some blackheads can nearly always be found. Conditions which may be mistaken for it are *rosacea* (p 409) and eruptions due to *bromides* (p 122) *iodides* (p 124) *chlorine*, *tar* and *mineral oils* (p 103) *Bromides* and *iodides* produce eruptions resembling pustular acne but without comedones on areas characteristic of acne e.g face and back. If however these eruptions are superimposed on existing acne, comedones will be present. *Chlorine tar* and *mineral oils* produce acneiform lesions including comedones, but on areas not usually affected by acne e.g penis scrotum and forearms as well as on the face.

**PROGNOSIS** Most sufferers from acne will "grow out of it sooner or later but it may well be so much later that their faces are permanently scarred and their matrimonial or other prospects permanently damaged. Not only so but quite a number of individuals become hypersensitive about their spotty appearance and develop an "inferiority complex" Seeing that it is exceptional to find a case of acne which cannot be cured or at all events greatly improved by suitable treatment, it always appears to me to be very cruel to tell an enquiring parent not to bother about her child's spots as she (or he) will "grow out of them."

**TREATMENT** I have not found that diet in this country makes any appreciable difference in the majority of cases except that eating chocolates generally makes the spots worse. The very oily cooking in some continental countries, however, does sometimes appear to aggravate acne in English girls and lack of sufficient vitamin C does likewise. Constipation and a sedentary indoor existence should be corrected if possible but one often sees quite severe acne in people who take a great deal of open-air exercise.

The first essential in treatment is plenty of soap and hot water to remove the excessive sebum. It is interesting that acne should begin so often in schoolboys and girls, who as a class are well known to have an aversion to soap and water. The next essential is the application of sulphur in some form. My favourite prescription is

R	Zinci sulphatæ	gr xxii	45
	Pot. sulphuratae	gr xx	4
	Acetone	℥ ii.	25
	Aq. rosae	℥ i.	100

Solve Ft. Lotion.

This can be increased in strength up to one drachm or more of the active ingredients to the ounce. This lotion should be applied two or three times a day after washing and drying the skin. It should be rubbed in with the hand not with cotton wool, until it is dry. Any remaining powder which is not rubbed into the follicles will be rubbed off. After a week or ten days the skin will feel tight and will be rough and peeling. The patient must be encouraged to continue the lotion once or twice a day and may be persuaded to do so by being given the following cream to be applied once a day.

R	Ung. ac. salicyl. (20%)	℥ i.	50
	Glycerinae amyl.	℥ i	50
	Ol. ros. geran.	q.s.	

In cases where this lotion does not seem to be successful a combination of resorcin and sulphur gr x. to ℥ ii. (2-25%) of each in an ounce of hydrous zinc ointment may be applied thickly to the face each night and washed off in the morning. A good plan is to start with about gr xl (8%) of each to one ounce and increase the strength of each drug by gr x (2%) every time one ounce is made up until the patient finds it becoming too strong. A vanishing cream base may be used instead of hydrous zinc ointment and in this case it should be rubbed in well until it disappears. X-rays must not be used at the same time as strong applications of this sort.

In some cases in girls and women hexoestrol 5 mg daily for two weeks starting on the third day of each monthly period, as suggested by Barber has appeared to be helpful if continued for some months. Alternately larger doses given for a short period together with ultra violet light, appear to augment its effect. The usual course is 5 mg of stilbonestrol daily in men and women, for a month. Unfortunately the doses of oestrogen required



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really to control acne are liable to cause serious uterine haemorrhage in women and to stop spermatogenesis in men, although this effect is probably only temporary. Consequently adequate oestrogen therapy is only exceptionally justifiable in acne.

Patients inclined to acne must be warned against using greasy creams on their faces. Their skins are much too greasy already, and additional grease of almost any kind seems to aggravate the acne.

Comedones should be methodically expressed with a comedo expressor—an instrument shaped like a small spoon with a hole in the centre. The difficulty is to find one with a small enough hole. The majority of those sold have holes so big that they are only useful for exceptionally large comedones such as sometimes occur on the back. The hole for the average comedo should just admit an ordinary-sized pin and the diameter of the spoon should not exceed 2 mm. The skin should be wiped over with ether, acetone or spirit and the hole of the instrument placed over the comedo. Pressure combined with gentle rocking will then cause the comedo to come out through the hole. After removal of comedones the zinc sulphide lotion should be applied. Girls should not be allowed to spend hours in front of a looking glass "squeezing spots" with their finger nails. In this way one form of "*acne excoriée des jeunes filles*" is produced. Pustular acne spots should be opened with a sterile needle and the pus wiped away with cotton wool moistened with spirit.

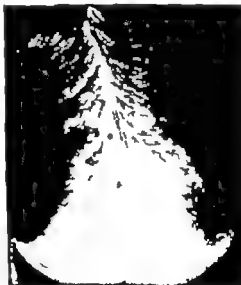
The indurated abscesses which occur in some cases of acne should be opened at their lowest points with a fine tenotome or cautery and the pus squeezed out.

Vitamin A given in doses of 200 000 to 300 000 units daily for 3 to 6 months, sometimes has a beneficial effect by virtue of its action on the hyperkeratinisation.

ULTRA VIOLET LIGHT treatment is useful in many cases of acne. The light should be given in biggish doses so as to cause some erythema and peeling of the skin. It is particularly useful in extensive cases of pustular acne on the back and chest but its effects are apt to be temporary.

X RAYS are of the greatest value in almost all cases of acne. They not only greatly accelerate cures by means of the lotion

mentioned above but they often make cure possible in cases in which it otherwise seems impossible. I always use as little X rays and as much lotion as possible in the treatment of acne. My usual practice is to give a dose of 50r of X rays once a week for two or three times and then to increase the dose to 100r and the interval to two weeks. A total dosage of 800r to each area is often required. In very pustular cases and in patients with sensitive skins it is better to use doses of only 50r and give more of them. If X rays are used along with the sulphur lotion or paste the patient must be warned to ease up the use of the latter if any redness of the face develops. If ultra violet light is used after a course of X rays care must be taken not to start it until a month has elapsed since the last X ray treatment and to give only small doses to start with, as the skin may redden more easily than normally and may even develop telangiectases later (Whitfield). Patients should also be warned of the similar risks of exposure to strong sunshine during and shortly after a course of X ray treatment. Cures obtained by the combination of X rays and lotion seem to be permanent provided that the patient continues to apply the lotion a few times a week for some months or until all tendency to relapse has vanished. There is a popular idea that X ray treatment causes or increases scarring. That this is not the case was shown by experiments by H D



Dr. Adams's Case  
FIG. 187  
Acne frontalis.

Niles,<sup>1</sup> in which in 40 patients with acne vulgaris only one cheek was treated by X rays and the final appearance of the two cheeks was compared. There was no difference between the scarring on the two cheeks in 33 cases, more scarring on the untreated cheek in 5 and more on the X-rayed cheek only in 3. If however excessive X ray treatment is given it may be followed later by some atrophy of the skin, with or without telangiectases.

Skin planing when carried out by an expert can remove much of the scarring caused by acne (p. 49).

**Acne frontalis**—**acne varioliformis**—**acne necrotica**. An eruption occurring on the frontal and temporal regions and characterised by yellow scabs embedded in the skin leaving depressed oval white scars when they fall out.

**ETIOLOGY** It seldom occurs under the age of twenty five and is much more common in men than in women. According to Sabouraud it is due to an infection of the pilo-sebaceous follicles with staphylococci. It generally occurs in patients with seborrhoea oleosa.

**PATHOLOGY** Inflammation of the pilo-sebaceous follicle and its immediate surroundings followed by necrosis. The epidermis grows underneath the slough so formed so that when the latter is thrown off a pit is left.

**SIGNS AND SYMPTOMS.** The early lesions are indurated reddish papules which rapidly undergo necrosis in the centre. The slough so formed is yellow or brown in colour and remains embedded in the skin for a week or two. When it falls out it leaves a small pit which eventually becomes a depressed white scar. The forehead, temples and sides of the head an inch or two above the ears are the usual sites. The neck and back are sometimes affected.

**PROGNOSIS** The disease is chronic and without treatment may last for years with periods of intermission. It is sometimes very resistant to treatment.

**TREATMENT** Topical antibiotics, such as 1% neomycin lotion, heal the lesions up quickly but they do not prevent new ones and treatment has to be prolonged. Salicylic acid sulphur

<sup>1</sup> Arch. Derm. and Syph., 1933 27 59.

ammoniated mercury resorcin and oil of cade in the form of ointments are all useful. In some cases X ray treatment brings about a dramatic cure.

**Rosacea**—*acne rosacea*. This disease is included here for the sake of convenience although it is only secondarily a disease of the sebaceous glands. Primarily it is a paralytic distension of the superficial blood vessels of the skin of the face a secondary hypertrophy of the sebaceous glands soon occurs and there may be numerous superficial pustules.

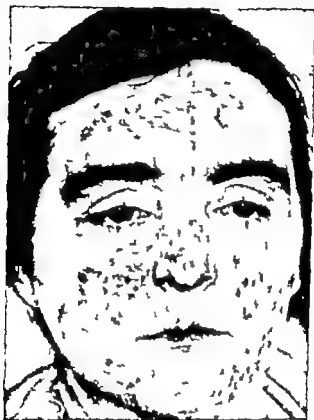
**ETIOLOGY** The disease seldom occurs before the age of thirty years it is more common in women than in men and is particularly common about the menopause, no doubt owing to the unstable state of the vasomotor system at that time. Certain factors, which tend to cause facial vaso-dilation in normal individuals, aggravate rosacea. Among these are heat, dyspepsia, hot or highly spiced foods, hot drinks, alcohol, severe menopausal symptoms, seborrhoea and emotional stress. None of these however play any fundamental part in the causation of the malady nor do they occur more frequently in individuals suffering from rosacea than in healthy persons. On the other hand the effect of both sunlight and cold winds on the disease is striking, the initial onset and subsequent relapses usually occurring in the spring or early summer. The majority of patients state that they have never been able to tolerate much sunlight the facial skin becoming easily burnt and failing to pigment.

**PATHOLOGY** Dilatation and new formation of capillaries in the upper third of the corium. Infiltration with small round cells and later with epithelioid cells and giant cells. This infiltration occasionally forms semi translucent brown nodules resembling the "apple jelly" nodules of lupus vulgaris. Degeneration of elastic tissue in the papillary layer. Hypertrophy of the sebaceous glands. Later the nodules of infiltration break down into pus and many collections of polymorphs are then found in the corium in all stages of degeneration. Scarring follows the healing of these abscesses.

**SIGNS AND SYMPTOMS.** For some months there may only be

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a tendency to flushing of the face after meals or hot drinks or in hot rooms. Later this becomes more or less permanent being most marked on the nose and the flush patches on the



*Dr. Archibald Gray, M.D.*

FIG. 152.

ROSEACNE.

cheeks the centres of the forehead and chin also are often affected. Dilated individual vessels can often be seen. The sebaceous glands hypertrophy and with the surrounding infiltration form red papules which may be confluent. Superficial pustules then appear in or alongside the hypertrophied glands. There are no blackheads. The scalp is often scurfy

or else shows signs of oily seborrhoea. In exceptional cases an enormous hypertrophy of the sebaceous glands of the nose takes place leading to the condition known as *rhinophyma* or cauliflower nose. This is commoner in men than in women. For eye affections in rosacea see p 413

**DIAGNOSIS.** From *acne vulgaris* (p 399) This occurs almost always before the age of thirty years and seldom lasts after that age, whereas rosacea seldom begins before twenty five or thirty. *Acne vulgaris* always exhibits some blackheads whereas rosacea never does. In a patient aged about thirty early rosacea and late *acne vulgaris* may however overlap. *Acne vulgaris* tends to affect principally the sides of the face, i.e. the temples, sides of the cheeks and sides of the chin whereas rosacea is most prominent on the nose, flush patches of the cheeks and centre of the forehead and chin.

From *Lupus erythematosus* (p 305) Here the red patches are sharply circumscribed instead of diffuse as in rosacea. There is usually some scaling and in older patches central atrophic scarring. In some of the patches the characteristic stippling caused by horny plugs filling the openings of the sweat and sebaceous glands can usually be made out. In *lupus erythematosus* the affected skin tends to be dry and atrophic except in the most acute cases, whereas in rosacea it tends to be greasy and hypertrophic-looking. Pustules are common on patches of rosacea, but very rare on those of *lupus erythematosus*.

From *Rosaceous tuberculide*. If a case of apparent rosacea does not respond to the treatment suggested below the possibility of its being one of the rather uncommon rosaceous tuberculide must be considered (p 217)

**PROGNOSIS** Rosacea is a chronic disease with little tendency to spontaneous cure. It is, however very amenable to suitable treatment most early cases can be cured and the later ones greatly ameliorated.

**TREATMENT General** The various possible aggravating factors must be treated if operative. Oestrogen therapy for

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menopausal symptoms is particularly helpful, 0.01 mg of ethinyl oestradiol being given two or three times a day in courses of 3 weeks with one week's rest in between. In cases which are markedly sensitive to sunlight, Chloroquine mg 250 two or three times a day is sometimes useful. Gentle sedation for a short period of time with amylobarbitone gr  $\frac{1}{2}$  2 or 3 times a day can be useful, but I have found psychiatric therapy disappointing.

The patient must avoid everything which tends to cause flushing of the face, such as sitting in front of a fire, coming into a hot room from the cold outside and so on.

**LOCAL TREATMENT** Sulphur appears to have a specific effect on the disease. It nearly always clears the papules and pustules and generally decreases or even abolishes the erythema. It is used in a 1 or 2% strength in calamine lotion or better still, in an emulsion base such as 30% ung emulsificans in water. The latter should be well massaged in twice daily the massage having a definite beneficial effect. The treatment should be persisted with for 6 to 8 weeks and frequently if continued thereafter daily or every other day will prevent relapses.

X rays in small doses, e.g. 80-100r once a week for three or four times, are of great assistance in treating many cases of rosacea though in some they only aggravate the redness. Individual dilated vessels may be obliterated by means of the galvanocautery or by electrolysis. Rubbing with  $\text{Co}_2$  slush (p 40) at fortnightly intervals can be very helpful in obstinate cases. A gauze pad is wrapped round the end of a tongue spatula and dipped in the slush. Liquid oxygen or nitrogen can also be used.

The treatment of rhinophyma is surgical.

**Eye affections in Rosacea** (see A J B Goldsmith *Brit Jour Derm.* 1933 65 448 and P Borne *ibid*, p 453) Blepharitis, conjunctivitis and marginal corneal ulceration often occur but rather in association with seborrhoea than with rosacea. True rosacea keratitis and ulceration affect the lower two-thirds of the cornea i.e. the exposed part and lead to corneal opacities and impaired vision. Keratitis occurs for the first time at the same time as the skin lesion in 51% of cases earlier in 70% and

later in 27%. It is liable to very frequent relapses, without relation to the condition of the face, especially in spring and early summer. The majority of cases, however, can be controlled partly or wholly with cortisone eye drops.

## DISORDERS OF THE SWEAT GLANDS

Individuals vary greatly in the amount of perspiration which it is normal for them to secrete. Some perspire freely all over on the slightest provocation while others practically never do so at all. The latter are apt to find hot weather very trying and I have a patient whose temperature goes up on a hot day presumably because of the lack of the cooling provided normally by evaporation of sweat. Cases are on record of congenital ectodermal defect, with absence or scarcity of sweat glands, in which the patients when working in hot weather had to have buckets of water thrown over them at regular intervals to keep down their temperatures.

Localised hyperidrosis—excessive sweating—commonly affects either the axillae or the palms and soles. In any of these situations it may be most trying to the patient, and some of them become very self-conscious as a result. In severe cases it may even interfere seriously with the sufferer's employment. Occasionally one meets with cases in which a single patch of skin, e.g. on the forehead, sweats excessively. The sweating in localised hyperidrosis tends to be produced more by emotion than by exertion.

**TREATMENT** Even the most severe cases can be cured by preganglionic sympathectomy but such a drastic treatment is hardly justified except in the worst ones. Hexa and penta methonium salts given by mouth abolish palmar sweating for some hours. Small doses should be given at first and increased gradually (C. A. Keele.) Ordinary cases can usually be cured or greatly improved by X rays. My usual practice is to give 300r once a month until the condition is nearly cured. Two, three or four treatments may be required. It is important to remember that hands or axillae are much more of a nuisance if too dry than if too moist, and that if the drying up process is overdone it cannot be altered. In the case of perspiring feet



correction of a flattened longitudinal or transverse arch sometimes has a very good effect. In cases where X ray treatment is not obtainable palliative measures must be taken. These consist in frequent bathing of the affected parts and the application of a 1-5% solution of sodium hexametaphosphate twice daily (Jones, Murray & Ivy *Industrial Medicine* 1937 8 459) or of salicylic acid two or three per cent. in bath eau de Cologne. Mopping on a 10% solution of formaldehyde (commercial formalin 1 part water 3 parts) is often effective but may set up dermatitis. In the case of perspiring feet the socks or stockings must be changed twice daily the feet bathed in potassium permanganate solution of a dark pink colour and after drying dusted over with some such powder as acid. salicyl gr xv., pulv. cretae gall. ad 3 l. (3%) some of this should also be shaken into the socks or shoes. Sodium hexametaphosphate also may be used as a dusting powder either by itself or 5% or 10% in talc powder.

**Bromidrosis**—offensive sweating—sometimes complicates hyperidrosis and is due to bacterial decomposition of the epidermis macerated by the excessive sweat. It most commonly affects the feet. The treatment is that indicated above for hyperidrosis with in addition the destruction of boots or shoes soaked in the offensive sweat and the correction of flat foot if present. In the German army during the first World War the application to the feet of chromic acid solution 5% once a week was found to be effective.

**Sudamina**—*miliaria crystallina*. A crop of minute glassy vesicles containing sweat occurring usually on the trunk in patients with acute febrile illness, e.g. following the crisis of pneumonia. The vesicles are due to lifting up of the horny layer by sweat which is unable to escape owing to obstruction of the duct. The eruption causes no symptoms and disappears in a few days. No treatment is called for.

**Dysidrosis** is a name given to certain rare cases of pompholyx (p. 330) in which the vesicles communicate with the sweat ducts and contain sweat like sudamina. (Tilbury Fox, Whimster)

## CHAPTER XX

### TUMOURS OF THE SKIN

Moles, vascular and fibromatous naevi, and sebaceous cysts are described in Chapter IV p. 50. Hair follicle naevi and sweat gland naevi cannot be described here. A larger text book must be consulted.

Warts and molluscum contagiosum are described in Chapter X, pp. 251-262. The keratoses due to chronic solar dermatitis (p. 81), arsenic (p. 117) tar (p. 103) and mineral oils (p. 104) are described on the pages indicated.

### INNOCENT TUMOURS

**Dermoid cysts**—"inclusion dermoids"—containing hair and hair follicles, sebaceous glands, etc. are sometimes found at the sites of the fusion of embryonic lobes, *e.g.* about the outer canthus, sides of nose, neck, and in the median raphe of the perineum and scrotum. Their treatment is surgical.

**Epidermic cysts**—milia—composed of small encysted accumulations of horn cells in the corium, are commonly seen as little white specks a millimetre or two in diameter on the upper parts of the cheeks and about the eyelids where they are often taken for sebaceous cysts. They are also seen on the scars of epidermolysis bullosa and occasionally on those of pemphigus, dermatitis herpetiformis, herpes zoster and injuries.

**Treatment.** They can be shelled out if the surface is incised, or they may be destroyed with a galvano-cautery or electrolysis needle.

**Lipomata** are tumours composed of adipose tissue. They may be of almost any size and occur anywhere. They are soft rounded swellings in the subcutaneous tissue which often fluctuate. The treatment is surgical.

**Cheloid. Keloid.** An irregular hypertrophy of the connective tissue of the corium forming a raised ridge or plaque usually on the site or scar of previous injury.

**ETIOLOGY** Apparently individual predisposition has a large share in the development of chaloids, some persons developing them after trivial injuries e.g. painting with iodine. Ordinarily



FIG. 189

Miba. Note situation and the absence of inflammation and comedo.

they are liable to occur on the scars of wounds which have remained open for a long time e.g. tuberculous sinuses, and those of burns and scalds. The so-called spontaneous chaloid is most common over the sternum where however it probably arises

originally from an acne pustule. Acne cheloid or syccosis nuchae arises around pustular acne on the nape of the neck (p. 196)

**PATHOLOGY** There is an immense hypertrophy of the collagen of the dermis, the bundles running mostly parallel to the surface. This hypertrophy of the connective tissue appears to begin around the blood vessels and extends along them beyond the visible limits of the growth. The elastic tissue is absent and, in the centre of the growth, the blood vessels also



Fig. 180

Hypertrophic (Cheloid) Scars on defoid region following burn.

**CLINICAL FEATURES.** Cheloids form ridges or plaques of irregular shape, often with claw like prolongations spreading into the surrounding skin. The colour of the growths is red purplish or white and the surface smooth and shiny. They are often painful and tender. They grow slowly and after reaching a certain size remain stationary or occasionally retrogress. A distinction is sometimes drawn between a true cheloid and a hypertrophic scar the difference being that the latter remains limited by the borders of the original scar while the cheloid invades the skin beyond these borders.

**TREATMENT** Young cheloids or hypertrophic scars yield

readily to repeated doses of  $\lambda$  rays e.g (400r) filtered through 1 millimetre of aluminium at monthly intervals. If more than two or three such doses are given telangiectases are



FIG 191

"Spontaneous" Cheloid over sternum.

likely to occur subsequently on the scar but patients seem to prefer these to the cheloids. Old cheloids are very resistant and should be excised X ray treatment being given before excision and repeated to the wound before the stitches are removed. *Excision without subsequent X ray treatment is always followed by recurrence* Cheloids resistant to X rays can sometimes be removed by the use of radon seeds inserted in the growth Small cheloids are sometimes much improved by freezing with carbon dioxide snow for 30 secs. or so Hydrocortisone (25 mg per m.l.) injected directly into the cheloid

at one to three week intervals on several occasions is another useful method of treatment

**Xanthomata.** Small tumours of a yellow or pinkish yellow colour containing quantities of a cholesterol fat. There are three varieties *Xanthelasma palpebrarum* or xanthoma planum in which yellow plaques appear in or just under the skin of the eyelids, especially the upper eyelid, in middle aged or elderly people, generally women These are best removed by very careful painting with liquid trichloroacetic acid after ringing with petroleum jelly The resulting scab comes off in two or three weeks leaving a very inconspicuous scar *Xanthoma tuberosum multiplex*, in which the tumours appear usually on the extensor surfaces of the larger joints but may be very widespread.

**Xanthoma diabeticorum** The lesions are similar to those of xanthoma tuberosum multiplex but are redder tend to appear more suddenly and occur in patients with diabetes, either young or old. Occasionally the lesions disappear if the diabetes is controlled, but not invariably.

For information on xanthoma a larger work on Skin Diseases should be consulted.



FIG. 102.

Xanthoma tuberosum multiplex. Elbow. Blood cholesterol 633 mg. per 100 c.c.

## MALIGNANT GROWTHS OF THE SKIN

The malignant growths which commonly arise in the skin are squamous-celled epithelioma or carcinoma, and basal-celled epithelioma, commonly known as rodent ulcer. Less common are various types of intra-epidermal epithelioma of which Paget's disease of the nipple is one while melanotic growths arising in pigmented moles are rarer still. Primary sarcoma of the skin is also very rare. Secondary deposits of carcinomata and sarcomata arising in other organs may occur in the skin as elsewhere.

Although the majority of the skin cancers fall fairly definitely into the squamous or the basal-celled groups yet there are a number of types in which the growth has histological and clinical characters intermediate between the two. Moreover a single growth may be typically basal-celled in one part and squamous- or prickle-celled in another. This corresponds

to the clinical observation that a growth which has increased slowly for several years and appears to be and is in fact a rodent ulcer may suddenly take on more rapid growth at one point. A section will show that the growth here has become squamous- or prickle-celled in type.

**Squamous-celled epithelioma** **ETIOLOGY** Squamous epitheliomata seldom appear before the age of forty years, and they are more common in men than in women. The majority of these tumours arise under pre-existing keratoses which in their turn may have been produced by *chronic solar dermatitis* (p. 80) or prolonged exposure to *tar* (p. 103) *arsenic* (pp. 100-119) or *mineral oils* (p. 103). Savatard, who has a very large experience of cancers of the skin, holds that such keratoses are malignant from the start.



FIG. 102.

Squamous-cell epithelioma of the lip. Duration 6 months.

Other conditions upon which squamous epitheliomata may supervene are old standing *lupus vulgaris* (p. 205) old X-ray or radium burns (p. 85) or the sites of *chronic irritation* or *repeated minor injuries*. Squamous epitheliomata are also a feature of the rare disease known as *xeroderma pigmentosum* (p. 81).

A small number of squamous epitheliomata arise apparently *de novo* and without any pre-existing lesion to account for them.

PLATE VIII



Squamous Epithelioma on *Lepus Vulgaris*





**PATHOLOGY** The growths consist primarily of a multiplication of the cells of the prickle-cell layer with a resulting enlargement of the interpapillary processes. The prickle-cells then break through the basal layer and invade the corium as columns of cells forming an irregular branching network in the lymphatic spaces. Near the skin surface the cells of these columns can still be differentiated into basal and prickle-cell layers, but deeper down this distinction is lost and the prickle-cells also lose their inter-connecting fibrils and become irregular in size and shape. They have large pale vesicular nuclei, and many of them exhibit irregular mitotic figures. Here and there in the masses of cells keratinisation takes place resulting in the formation of spherical bodies composed of concentric layers of horn cells and known as cell nests. About the columns of invading cells there is an infiltration of round cells, plasma cells, and mast cells, which represents a defensive reaction on the part of the skin.

**SIGNS AND SYMPTOMS.** Squamous epitheliomata when arising *de novo* appear first as small thickenings of the epidermis with a slight adherent scale on the surface. In a week or two a definite nodule has appeared which may reach a diameter of half an inch in three weeks. There is no sign of inflammation, the growth is firm opaque, and the colour of normal skin. Later the surface may become broken and possibly infected and signs of inflammation appear in and about the growth. When a squamous epithelioma arises under a pre-existing keratosis the latter appears to increase in area and to become more prominent while signs of the growth may be detectable at the edges. The patient may complain of its irritating more than usual.

Squamous epitheliomata tend to invade lymphatic glands and to cause metastases, although they vary a good deal in the rapidity with which they do so. Squamous epitheliomata may arise anywhere on the skin, but are most common on the face and hands and at muco-cutaneous junctions, e.g. lips, nose and anus, also on the penis and scrotum. The latter site is the usual one in chimney sweeps and in "mule-spinners" owing to

irritation of the scrotal skin over long periods by soot and mineral oil respectively

**DIAGNOSIS.** The diagnosis of early squamous epithelioma of the skin has to be made from *kerato-acanthoma* (p. 423), *basal celled epithelioma* (p. 424) *molluscum contagiosum* (p. 213), *sebaceous cyst* (p. 70) *mole* (p. 62) *wart* (p. 231) and *boil* (p. 197). The diagnosis is not usually difficult if squamous epithelioma is kept in mind as a possible alternative to the above diagnoses. In many cases of keratosis it is very difficult to say when a given lesion is becoming malignant. When in doubt



FIG. 194

Rodent ulcer of inner canthus Duration 14 years.

excise is a good rule here. On the lip nipple and anus especially the possibility of *primary syphilis* should never be forgotten (p. 219).

**PROGNOSIS** As in all forms of cancer the prognosis should be guarded. It is grave if lymphatic glands are already affected or if secondary growths are present. If seen early while the growth is

only half an inch or less in diameter and if promptly treated the prognosis is good. Savatard for example has excised a large number of early carcinomata of the skin and has not had a single recurrence.

**TREATMENT** In the early stages local excision only is necessary a margin of a quarter of an inch or so all round and beneath the visible and palpable limits of the growth being all that is required. If there is any suspicion that the growth has invaded the lymphatic glands, these should be removed or treated with X rays or radium but in the early cases this is seldom necessary. Instead of excising it the tumour may be treated equally well by radiotherapy although squamous

epitheliomata are less radio-sensitive than are the basal-celled variety

Keratoacanthoma (Freudenthal) Molluscum Sebaceum (MacCormac and Scarff), Molluscum Pseudocarcinomatousum (Lan



FIG. 105

Keratoacanthoma. Duration 6 weeks

cer) These tumours occur a little more commonly in men than in women and at a slightly earlier age than squamous-cell epitheliomata. The majority of the latter occur between the ages of 60 and 80 while most kerato-acanthomata are seen between the ages of 50 and 70. The commonest sites are the cheeks, nose and fingers and hands, but they also occur elsewhere on the face neck and the scalp and rarely on the limbs and trunk. They almost always occur singly as firm hemispherical nodules reaching a diameter of 1 to 2 cms. in four to five weeks. The sides of the lesion which are reddish to skin coloured rise abruptly from the surface of the skin. The summit is usually covered by a crust of keratin which, when removed reveals a crater. The majority of lesions continue to grow for 4 to 6 weeks the crust then separates and gradual flattening occurs over the next month or 6 weeks, the final appearance being a rather unpleasant puckered scar. Occasionally however growth continues for longer and large lesions are formed which take three months or more to resolve.

Histologically there is marked acanthosis of the prickly-cell layer tongues of which run down in a convex manner into the dermis. A mass of keratin lies on the surface and penetrates the centre of the tumour. The prickles are intact and there are very few mitoses but the basal-cell layer is poorly defined and partially obscured by a dense infiltrate of polymorphonuclear leucocytes and lymphocytes in the underlying dermis.

Although these tumours are self healing they are best treated by curettage and cautery as in the case of a wart (p. 239), since a better cosmetic result is obtained in this way.

Kerato acanthomata are slightly less common than squamous-cell epitheliomata. Very rarely one of them develops into a true epithelioma and they may be regarded as failed epitheliomata, the reason for their sudden cessation of growth after a few weeks being at present obscure.

Basal-celled epithelioma.—rodent ulcer. **ETIOLOGY.** The majority of basal-celled epitheliomata appear to arise *de novo* but in Australia at all events they frequently develop under solar keratoses. In this country the latter tend to give rise

to squamous epitheliomata. Basal-celled epitheliomata are common in both sexes after the age of forty but cases have been recorded occurring as early as twelve (Sequeira) and I have had several beginning between twenty and thirty. The vast majority of them occur on the face especially between the levels of the upper lip and the eyebrow but they may arise anywhere on the skin surface.



FIG. 184

Rodent Ulcer. Note Rolled Edge.

These tumours appear to develop either from the basal layer of the epidermis or from the outermost layer of the hair follicle. They consist of ramifying processes made up of cells resembling those of the basal layer of the skin. That is to say they have relatively large and deeply staining nuclei, and at the surface of each portion of the growth they are arranged as an unbroken layer of columnar cells. Around the growth the fibrous tissue of the corium is condensed and there is an infiltration of round cells and plasma cells.

SIGNS AND SYMPTOMS. The earliest lesion is a small semi-translucent pearly papule which may irritate a little. The

patient probably scratches it and a small crust forms. This may get picked off at intervals but always re-forms and the lesion never really heals. From this stage the growth may develop

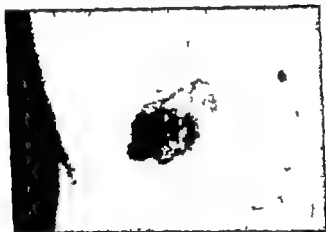


FIG 107

Rodent ulcer Superficial Type. Duration ten years.

in various ways. (a) The *ordinary type* With a raised semi translucent pearly border the so-called "rolled edge" sometimes traversed by visible venules, and with a shallow ulcer covered by a crust in the centre (b) The *superficial cicatricial type* This is most common on the temples and scalp also on the trunk, where it is often multiple. It appears as an irregular

scar with small crusts here and there and a very slightly raised pearly border. It tends to progress slowly remaining quite superficial and scarring up in the centre while advancing at the edge. This type has to be diagnosed from *lupus vulgaris* (p. 203), and from *tertiary syphilis* (p. 261). (c) The *card-like type*. This forms infiltrated plaques in the skin which may have to be distinguished from *morphea* (p. 436). It has, however, a much greater tendency to ulceration, while careful examina-



Dr Hugh Gordon's Case.

FIG. 108.

Rodent Ulcer. Card-like Type. Lesion. Section showed change to squamous epithelioma in central part.

tion with a lens will probably reveal a very narrow pearly border. (d) The *cystic type*. This forms bluish semi-transparent cysts from an eighth to half an inch in diameter with a smooth surface and containing a gelatinous fluid. (e) The *cerebral type*. This is fortunately uncommon. It is characterised by comparatively rapid growth with immediate ulceration which extends deeply and invades all tissues impartially leading to a deep excavation of the nose or orbit. This type is usually eventually fatal from haemorrhage or septic pneumonia.

Rodent ulcers are very often multiple. They never invade lymphatic glands except accidentally by direct extension, nor



do they give rise to metastases. They are however "locally malignant" invading and destroying other tissues and having a strong tendency to recur unless completely removed or destroyed.

**DIAGNOSIS.** Any persistently crusted spot on the face of an elderly person should be suspected of being a basal-celled epithelioma and be examined from this point of view.

**Diagnosis from squamous epithelioma (p. 420)** This grows

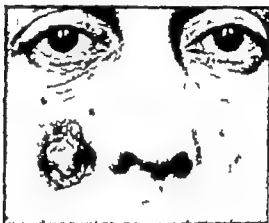


FIG 129.

Rodent ulcer : This proved to be one of the deeply penetrating type.

much faster and may reach a diameter of half an inch in three weeks, while the rodent ulcer has generally been present for some years before the patient bothers to take advice about it. The squamous epithelioma tends to have a convex surface at least in the early stages, whereas the rodent ulcer very rapidly breaks down in the middle and so gets a depressed centre with a raised pearly edge. The squamous epithelioma tends to be whiter and more opaque than the rodent which is rather pearly and translucent. The squamous epithelioma tends to have a scale in the centre the rodent a crust.

From *molluscum contagiosum* (p. 212). These growths are nearly always multiple are most common in children, and

consist of small pinkish or pearly tumours with a surface which is shiny except for a single dull spot near the centre. If



FIG. 200

Radent Ulcer over sternum. Duration 7 years. Note at top and bottom outlying areas of growth probably intra-epidermal

incised a caseous mass can be expressed leaving a thin shell of epidermis.

From tertiary syphilis (p 761) Here the ulceration is

usually more rapid the edge of the ulcer is sharp and punched out and the semi translucent pearly border is absent. In tertiary syphilis the Wassermann reaction is positive in 80% of cases, and the effects of anti-syphilitic treatment are usually rapid.

From *lupus vulgaris* (p 205). Here the disease has usually started in childhood or early life apple-jelly nodules are present and there is no pearly border.

**PROGNOSIS.** The prognosis in a case of rodent ulcer if seen early is good. Even extensive rodent ulcers can often be cured provided they are superficial. In cases where deep ulceration has occurred however the disease is very difficult to cure, especially if bone is invaded.



FIG 201

Paget's disease of the nipple. Duration 1½ years. Patient aged 62.  
Note disappearance of nipple. Contrast with eczema Fig 146

**TREATMENT** Excision carried a sixth to a quarter of an inch all round and below the extreme limits of the growth is usually successful. Owing to the frequent occurrence of rodent ulcers about the orbit such free excision, without subsequent deformity is often impracticable deformities may however be remediable by plastic operation.

Rodent ulcers are extremely radio-sensitive so that the treatment of choice is by X RAYS. The average dose is 3000r pre-

ferably given in daily fractionated doses for 8 to 10 days. Beta radiation, in the form of radio-active isotopes, may be used on superficial lesions, especially the multiple type occurring on the trunk.

Other treatments are by curetting followed by the application of chromic acid, of trichloroacetic acid, or of even  $\text{CO}_2$  snow.

**Intra-epidermal epithelioma.** Sometimes the neoplastic epidermal cells instead of growing downwards into the corium grow upwards and sideways and remain, often for years, within the epidermis. The clinical appearance resulting is a very slowly extending plaque of which the colour is brownish or red and the surface either peoniasiform, with some scales or crusts, or else velvety or raw looking. Eventually a certain proportion of such growths may begin to grow rapidly invade the corium and come to resemble ordinary epithelioma. Examples of different types of intra-epidermal epithelioma are Bowen's "precancerous dermatosis, Quéyrat's érythroplakie" and Paget's disease of the nipple.

Treatment is by excision, X rays or radium.

**Paget's disease of the nipple** is a rather uncommon form of intra-epidermal carcinoma of the skin associated usually with an underlying duct carcinoma. It appears usually as a red glazed infiltrated plaque occupying the site of the nipple and areola, though sometimes the lesion is scaly and peoniasiform. It has to be distinguished from eczema (p. 322) and from primary sores of the nipple (p. 249). The principal points in the diagnosis from eczema are

	Eczema.	Paget's disease.
Age.	Any age.	After 40.
Side.	Often bilateral.	Unilateral.
Progress.	Gets better and worse.	Slowly but steadily worse.
Edge.	Often ill defined.	Well defined.
Surface.	Vesicular or crusted or scaly	Bright red, but may be dry with a few white scales and resemble psoriasis.
Induration.	None.	"Like a penny wrapped in flannel" but sometimes very slight.

Nipple.	Not retracted	Soon becomes flattened or retracted
Biopsy	No round bodies.	Round bodies," i.e. degenerated prickle cells, seen in scrapings and sections.

The treatment consists in removal of the whole breast. Paget's disease may occur elsewhere, e.g. umbilicus vulva perinaeum, etc.

*Malignant melanoma* arises most commonly in a dark brown or blue-black mole usually non hairy and flat or only slightly raised, situated in descending order of frequency on the lower limb head, neck or face, trunk or upper limb. Malignant change in a mole is suggested by increase in size or in pigmentation by irritation, pain or induration, or by enlargement of the regional lymph nodes. Such change may follow trauma or be associated with puberty or pregnancy. The treatment of malignant melanoma is a matter for the surgeon and includes wide excision of the growth and of the regional lymph nodes. Radiotherapy is contraindicated. The prognosis is gloomy.

## CHAPTER XXI

### ATROPHY AND SCLEROSIS

**Atrophy** of the skin may be the result of pressure, wasting disease interference with nerve supply previous inflammation, or senility. In addition certain types of "primary" or idiopathic atrophy are described whose cause is unknown.

Atrophic skin is thin, either smooth and glossy wrinkled like crumpled tissue paper or finely scaly. It may be white or grey or else bluish or dusky red, owing to its transparency and the atrophy of the subcutaneous fat allowing one to see the blood vessels. It is inelastic, so that if pinched up the fold takes an appreciable time to flatten out again. The hair on atrophic skin may turn white or fall out and the sweat and sebaceous glands share in the atrophy.

**Striae atrophicae** These are commonly seen on the abdomen and breasts of women who have borne children, but are also seen in men who have put on weight rapidly and sometimes about the upper arm in muscular individuals. They also occur in wasting diseases such as typhoid. The mechanism of their production appears to be that the rupture and destruction of the "elastic fibrils, which normally limit the extension of the collagen fibres of the dermis, allow the latter to extend far beyond their proper limits with consequent damage and subsequent repair by scar tissue. The destruction of the elastic fibrils may be due to over stretching (pregnancy) or the result of toxins or malnutrition (typhoid fever).

Striae atrophicae are an inch to several inches long, about a quarter of an inch in diameter and at first are reddish or purplish in colour. Later as scar tissue develops in them, they turn white.

**Senile atrophy** is characterised by thinning of the corium and epidermis, atrophy of the sebaceous and sweat glands, development of telangiectases or angiomas, and increased pigmentation

which is apt to be patchy. It is sometimes accompanied by pruritus (senile pruritus).



FIG. 202  
Scleroderma. Atrophic Type.

In some cases of scleroderma (p. 133) atrophy may take place following or instead of the thickening (Fig. 202).

In rare cases of syphilis a macular atrophy occurs. Here the

atrophic macules are bulged outwards by the pressure of the underlying fat.

**Primary atrophy** *Idiopathic macular atrophy* This usually occurs in young women and is characterised by the presence of atrophic patches round or oval in shape and from  $\frac{1}{4}$ -1 inch in diameter. In colour they are white or else bluish, owing to their transparency and they may be either level with the skin, depressed, or bulged out by the underlying fat. They are most common on the shoulders and the back and sides of the thorax.

*Idiopathic diffuse atrophy* (*Acrodermatitis atrophicans chronica*.) This usually affects women of about forty years of age. It generally begins on the dorsum of the foot with well-defined erythematous patches, red or bluish in colour. These gradually extend and new ones appear until the whole lower limb may be involved. The upper extremity may also be affected from the hands to the shoulder. In the later stages the skin is red, blue, or brown in colour atrophic and inelastic easily wrinkled, and transparent, so that the underlying veins and even tendons can easily be seen.

Probably both these types of "primary" atrophy are really secondary to some inflammatory process of unknown causation. No treatment has any effect on atrophy of the skin.

## SCLERODERMA

A group of affections of unknown origin in which the affected skin and subcutaneous tissue become thickened tough and ultimately atrophic.

**ETIOLOGY** Unknown. Numerous attempts have been made to incriminate the sympathetic system and/or certain of the ductless glands e.g. the thyroid. Scleroderma may however occur in association with either excess or defect of thyroid secretion or with a normal thyroid. It is most common in women and in early middle life.

**PATHOLOGY** Endo- and peri-vasculitis affecting both arteries and capillaries, with round-cell infiltration about the affected vessels in the early stages. Later occlusion of many of the vessels, disappearance of the cell infiltration, and great hyper



trophy of the white fibrous tissue of the corium and subcutis. The elastic tissue may be normal or may show all stages of degeneration down to complete disappearance. The hair follicles and the sweat and sebaceous glands appear to get strangled by the white fibrous tissue and consequently atrophy. The papillae are flattened out and the epidermis becomes thinned and atrophic. The sclerotic process may involve the underlying muscles also. Atrophy may occur instead of or following the hypertrophy of the white fibrous tissue.

Scleroderma occurs in three principal forms, generalised scleroderma and morphea, progressive systemic sclerosis (sclerodactyly, acrosclerous) and guttate scleroderma (lichen sclerosus vel atrophicus).

**Generalised scleroderma.** The onset is usually insidious, and the upper part of the body (neck or shoulders) is first attacked. The disease gradually spreads more or less symmetrically until after months or years the greater part of the body surface is involved. The affected skin is smooth, glossy, hairless, thickened, and resembles a bladder filled with hard ". Sometimes it is pigmented yellowish brown, in a diffuse or patchy manner. The affected skin is bound down to underlying structures and is consequently readily injured. Injuries are apt to result in chronic ulcers. Pressure atrophy of the muscles and bones with fixation of joints, deformity and contractions follows. The rigidity of the skin and involvement of muscles interferes with eating, swallowing, respiration, and movements of viscera. Death finally ensues from progressive malnutrition or intercurrent disease. Surprising recoveries occasionally occur.

The only visceral lesion which occurs in this type is a myositis in the muscles underlying the affected skin. The condition probably represents a generalized form of morphea and is thus distinct from progressive systemic sclerosis.

**Morphea (localized scleroderma)** (Fig. 203). This is much commoner than the generalised type. It appears as plaques an inch to a few inches in diameter or as bands many inches in length. The plaque may be preceded for weeks or months by an itchy patch of red or violet tint. This fades and is replaced by an indurated

plaque with an ivory-coloured centre and a lilac border. Usually the affection is not noticed by the patient until the sclerosis of the patch is evident. When fully developed the centre may be the colour of old ivory bluish, mottled or pigmented. The sur-

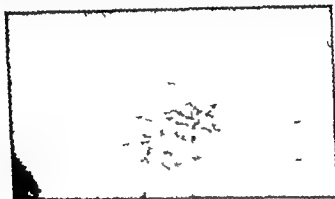


FIG. 203.

*Scleroderma. Morphoea. Plaque Type. Shoulder*

face is smooth and shiny and may be traversed by dilated capillaries. The affected skin cannot be picked up. This central area is surrounded by a lilac-coloured border  $\frac{1}{2}$ - $\frac{3}{4}$  inch wide whose colour is due to dilatation of blood vessels. In shape the plaques may be oval or irregular and they may be raised above level with or depressed below the general surface. They may occur anywhere but are usually situated on the neck, breasts (in women), chest, shins, or thighs, and are often symmetrical. The mucous membranes may be affected.

The band type of scleroderma is much less common than the plaque variety. It appears as bands chiefly on the limbs  $\frac{1}{2}$ - $\frac{3}{4}$  an inch or more in width and extending from a quarter to the whole length of the limb (Fig. 204). Bands may also occur on the trunk apparently following the distribution of posterior nerve roots (Fig. 205) or on the forehead in the distribution of the supraorbital nerve and may have to be distinguished from scars left by Herpes zoster. Plaques may or may not be present elsewhere.

**PROGNOSIS** The plaques extend slowly remain stationary for

months or years, and then retrogress with or without treatment. The affected skin may be restored to normal or may be indicated permanently by a pigmented or atrophic area (Fig 902).



FIG. 204

Scleroderma. Band Type.  
Right upper arm.



FIG. 205

Scleroderma. Band Type. Trunk.  
Note resemblance to scars of Herpes zoster

**Sclerodactyly — Acrosclerosis.** (progressive systemic sclerosis.) Raynaud's phenomenon usually precedes the onset but may accompany or follow it. The cutaneous change is first seen in the finger tips whence it spreads slowly up the fingers and hands.

It may also appear in the toes in the same way. The skin becomes tight stretched and hard, bound down to the underlying tissues, smooth and glazed. Ulceration of the finger tips, and

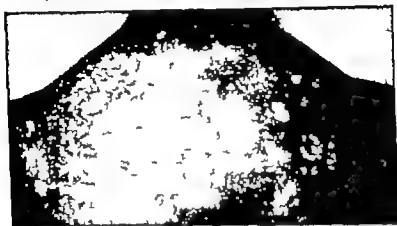


FIG. 206.

*Lichen sclerosus vel atrophicus (guttate scleroderma)*

patchy pigmentation and depigmentation are also common. Telangiectases appear especially on the face, the skin of which becomes stretched and taut, but not hard, the lips being thin and contracted and the nose pinched.

Scleroderma is a systemic disease. The lower end of the oesophagus is nearly always involved, leading to dysphagia. Other structures which may be affected include the rest of the gastrointestinal tract, the muscles, joints, kidneys, heart and lungs. Death usually ensues in a few years on account of cardiac or renal involvement, cachexia or intercurrent infection.

*Lichen sclerosus vel atrophicus (guttate scleroderma).* A chronic condition of the skin characterised by white sclerotic papules or atrophic macules from a half to a few centimetres in diameter at first discrete but sometimes eventually coalescing to form white atrophic plaques with irregular edges. The papules and macules are sometimes studded with dark horny plugs in the follicular openings and are sometimes surrounded by a narrow erythematous halo. The disease is most common in

middle-aged women and may occur on the neck, axillae trunk or limbs but is sometimes limited to the vulvar and anal regions where it may cause intractable pruritus and sometimes atrophy resembling kraurosis.

**TREATMENT OF SCLERODERMA.** There is no specific treatment. Sufferers from the generalised disease should be kept warm, have Turkish baths, and be massaged with warm oil. Their general health must be kept up as much as possible by good food and hygienic surroundings.

Thyroid gland by the mouth often seems beneficial. Pituitrin and pancreatic extracts as well as vitamins A and D have been recommended. Andrews advises dihydrotachysterol, a close watch being kept on the serum calcium. Cortisone has proved disappointing. In the localised form massage with warm oil X rays (100r doses) and ultraviolet light baths seem to be the most useful measures.

**Vitiligo or leucoderma** is the name given to a condition in which patches of skin lose their pigment and become perfectly white though no other changes take place in them. The melanoblasts in the affected areas have lost their power of forming pigment and give a negative dopa reaction. Vitiligo may occur in either sex and at any age. The white patches may appear on any part of the skin but are commonest on the face and neck, hands and wrists, lower abdomen and thighs (Fig. 207). They may be of any size or shape and are usually though not always, roughly symmetrical. They slowly increase in size until large areas of the skin are completely decolorised and the small remaining patches of normal skin may then be mistaken for pigmented areas. This mistake may be avoided by remembering that the vitiligo areas have convex margins and the normal areas therefore have concave ones. When vitiligo occurs on a hairy area such as eyebrows or pubes the hairs on the white patch may become white also (leucotrichia). The depigmented areas are sometimes surrounded by an excess of pigment in the immediately adjoining skin but this appearance is often illusory and the result of visual contrast. Vitiligo is most noticeable in the summer when the normal skin is tanned.

by the sun. The white areas having no protective pigment are easily made red and sore by exposure to sun or artificial ultra violet light. Vitiligo sometimes disappears spontaneously after months or years but more usually the condition spreads slowly



FIG. 207

Vitiligo. Note hyperpigmentation surrounding lesions on knuckles.

and may eventually involve nearly the whole of the skin. The cause of vitiligo is quite unknown and there has hitherto been no satisfactory treatment but the white patches may be disguised by staining with potassium permanganate solution or preferably with fresh walnut juice which gives a more natural brown colour

A treatment which has been used for some centuries in Egyptian folk medicine has in the last few years, received notice in western literature. It depends on extracts of a common umbelliferous plant *Ammi Majus*, Linn. These extracts contain three crystalline active principles which are furo-coumarin derivatives. One of these, *ammidin* is identical with *xanthotoxin* or 8-methoxypsoralen and has photosensitizing effects. The treatment is on the following lines (Sulzberger and Baer *Year Book of Derm.*, 1953-4 p 29 et seq) (1) One tablet of 75 mg of *xanthotoxin* is taken two to four times daily after meals. Side effects are nausea, insomnia and nervousness. (2) A 1% solution of *xanthotoxin* in alcohol is applied to the areas of vitiligo an hour before exposure to light. (3) A cream containing 15% para aminobenzoic acid is applied to the normal skin around the areas of vitiligo. (4) Exposure three to six times weekly to sunlight, carbon arc or ultra violet light. The exposures should be extremely short at first, three to five seconds per treatment, as severe bullous and febrile reactions may occur. The treatment has proved extremely disappointing in most Europeans, in whom it has no effect but in dark skinned races, and especially in negroes, complete though only temporary repigmentation may occur

## CHAPTER XXII

### VESICULAR AND BULLOUS ERUPTIONS

The bullous forms of Impetigo (pp 183 186) Erythema multiforme (p 293) Lichen urticatus (p 303) and Syphilis (p 269) are described elsewhere

**Dermatitis herpetiformis.** A disease characterised by erythematous vesicular or bullous lesions and severe itching which has to be differentiated from pemphigus vulgaris.

**ETIOLOGY** Unknown. It is most common between the ages of twenty and forty Both sexes are equally affected.

**PATHOLOGY** Oedema of the corium and epidermis leading to vesicle formation, dilatation of the vessels and perivascular infiltration with small round cells and eosinophils. According to G. H. Parnall and P. W. Hannay the bullae are always situated between the epidermis and corium (cf pemphigus p 146) The fluid in the bullae contains eosinophils, and the blood also shows an eosinophilia in about half the cases. This may vary from 4% to 40%.

**SIGNS AND SYMPTOMS.** The characteristic features of Dermatitis herpetiformis are the polymorphism of the eruption (erythematous blotches, grouped vesicles or bullae) and the itching. The disease tends to occur in attacks lasting weeks or months, with intervals of freedom over a period of years up to ten or fifteen. The general health is little affected except as a result of sleeplessness due to irritation or of secondary infection. Any part of the body may be affected but the mucous membranes are involved in only about 20% of cases. The lesions are patches of erythema which may be circinate and, arising on these groups of small vesicles resembling those of herpes, or bullae. The vesicles are always scratched and may become secondarily infected. They leave scars which are usually pigmented.

**DIAGNOSIS** This may be very easy or very difficult. The



polymorphism of the eruption and the intense itching burning or pain should suggest this disease, and if a pronounced eosinophilia is found in the blood and in the contents of the bullae it will tend to confirm this diagnosis. The principal points of distinction between *Dermatitis herpetiformis* and *Pemphigus* are

	<i>Dermatitis herpetiformis</i>	<i>Pemphigus vulgaris</i>
Eruption.	Multiform. Erythema, vesicles, bullae. Vesicles arise on previously red area. Vesicles generally less than two centimetres in diameter	Uniform, bullae only Bullae arise on normal skin. Bullae often several inches in diameter
Distribution.	Lesions tend to be grouped. Mucous membranes affected in only 20% of cases.	Lesions not grouped. Mucous membranes generally affected.
Subjective symptoms.	Irritation very severe.	Little or no irritation.
Eosinophilia in blood.	Tends to be higher (12-30%) and more frequent (50%)	Tends to be lower and less frequent.
Eosinophilia in vesicular fluid	Present.	Absent.
Cells scraped from floor of fresh bulla.	Not degenerate	Characteristic degenerative changes (Tsack).
Iodides	Aggravate eruption	Little effect.

The iodide test may be carried out by applying a 20% sodium or potassium iodide ointment for 24 hours to a part of the eruption.

The bullous forms of *impetigo* (pp 183-186) *erythema multiforme* (p 293) and *lichen urticatus* (p 303) also patchy vesicular eczema (p 320) may have to be considered in arriving at a diagnosis.

**Prognosis.** The attack may be mild and may only recur once or twice but in the majority of cases the disease lasts for years in recurring attacks with intervals of freedom. It is seldom fatal except indirectly by undermining the patient's health and many patients eventually recover.

**TREATMENT** In severe attacks the patient should be kept in bed. Diet should be light. Alcohol should not be given.

*Internal treatment.* Dapsone (diamino-diphenyl-sulphone) is the treatment of choice. It is given by mouth the dose being



FIG. 208.

*Dermatitis herpetiformis.*

50 mg., one to four times a day the majority of cases being controlled by 100 mg daily. The requirement varies from time to time owing to the natural remissions and relapses of the disease.

and patients can be allowed to regulate their own dosage. The effect is dramatic and rapid all signs of the disease disappearing in one to two weeks if the dose is adequate. Treatment, however must be continued indefinitely since it is suppressive and not curative. Toxic effects are infrequent. A depression of red cells and haemoglobin may occur in the first few weeks, but generally rights itself. Very occasionally a toxic hepatitis occurs, also early in treatment, necessitating discontinuing the drug.

Sulphapyridine but no other sulphonamide has a similar rapid and specific effect. The average daily dose is 1 or 1.5 grms. given in divided doses. Chronic listlessness and gastrointestinal symptoms are relatively common but leucopenia or agranulocytosis are rare and again usually occur in the first few weeks of treatment. Arsenic is now seldom used because of its toxic effects when taken over a prolonged period of time (p. 119). It is given as liq arsenicals 1 to 10 minims daily and also has a rapid suppressive effect.

*External Treatment* This is seldom necessary but simple antipruritics may be employed if the eruption is not completely suppressed by systemic treatment. Such are 1% phenol in calamine lotion and Eurax ointment or lotion. Hydrocortisone ointment is also effective.

*Hydroa gravidarum* is dermatitis herpetiformis occurring in pregnancy. When it occurs it does so usually between the third and sixth months of pregnancy sometimes appearing later in each successive pregnancy and occasionally only after parturition. The treatment is the same as for dermatitis herpetiformis, but is sometimes ineffective and steroids must be used. These suppress the condition in fairly low doses and do not have an adverse effect on the foetus. Quinine sometimes seems to cause improvement in this form of the disease but its tendency to hurry on labour must be remembered.

*Pemphigus vulgaris* A chronic and potentially fatal disease characterised by the appearance of bullae upon apparently normal skin.

**ETIOLOGY** Unknown. It is most common in patients over

forty but may occur at any age. Males are affected three times as often as females (Rook).

**PATHOLOGY** Dilatation of vessels and oedema of both corium



FIG. 200.

Pemphigus vulgaris. Note affection of flexures, lips and conjunctiva. The lesions are somewhat obscured by calamine lotion.

and epidermis. The cells of epidermis degenerate and liquefy forming bullae which are usually situated in the thickness of the epidermis, not between the epidermis and the corium as in

*dermatitis herpetiformis* (p. 443) The degenerated cells can be found abundantly in scrapings from the floor of a fresh bulla. They are rounded, the nuclei are spherical and only slightly coloured nucleoli are present. The cytoplasm which is abundant, basophilic and relatively clear round the nucleus becomes more dense at the periphery of the cell forming a very distinct dark blue areola (Txanok.) Post mortem changes which have been described in the central nervous system and other viscera may equally be seen after death from any chronic wasting disease.

**SIGNS AND SYMPTOMS** The bullae may appear first on the conjunctiva, the lips in the mouth or on the skin of the trunk or limbs. They are usually tense and arise on normal looking skin, i.e. without previous erythema. Adamson however states that if a patient is very carefully watched a transient erythema may be seen shortly before the appearance of a bulla. The contents of the bulla is at first sterile, clear serous fluid, but later it becomes infected and purulent, and a red areola then forms around the bulla. There is no excess of eosinophil cells in the contents of the bullae. The bullae may be of large size and keep on appearing in new situations. When they rupture they leave raw areas which are very tender to the touch these may eventually extend over a large proportion of the skin surface. The smaller bullae may dry up and form crusts.

*Nikolski's sign.* If the tip of the observer's finger is pressed on the skin of a patient with well developed pemphigus or dermatitis herpetiformis, or epidermolysis bullosa, the horny layer of the epidermis will be felt to slide on the underlying layers and may even be pushed off.

The mouth of a patient with pemphigus is apt to become extremely painful and septic on account of the loss of the epithelium from a large part of its surface. Other mucous membranes e.g. of the upper air passages, or vulva, may also be affected. The conjunctivae may be involved leading to adhesion of the lids, followed by "essential shrinkage" of the eyes.

The patient's general condition slowly or rapidly deteriorates there is pyrexia which may be increased by septic infection of the mouth and the skin surface there may also be albuminuria

vomiting, and diarrhoea. Patients sometimes recover in a few months from comparatively mild attacks but they tend to get relapses, one of which eventually proves fatal.

**DIAGNOSIS.** It should be remembered that pemphigus vulgaris is a comparatively rare disease. Bullous eruptions which may be confused with it are *dermatitis herpetiformis* (p 443) *erythema multiforme* (p 289) *bullous impetigo* (pp 183 186) *bullous toxic eruption* (p 121) *bullous reaction to moccasin bite* (p 153) and *acute malignant pemphigus* (p 451). The characteristic degenerated cells (p 448) described by Civatte, Piérard Dupont, Tzanck and others which can be found in scrapings from the floor of a fresh bulla of pemphigus are of considerable help in diagnosis, for they are not found in other bullous eruptions. (Rook, A. J., Whimster I W *Brit. Jour. Derm. & Syph.* 1950 62, 443) It must, however be stated that while classical cases of *dermatitis herpetiformis* and *pemphigus vulgaris* are easily distinguished many cases occur in which the differential diagnosis is impossible without prolonged observation. Moreover in some cases with lapse of time one disease appears to change into the other.

**PROGNOSIS.** Steroid therapy has completely changed the outlook in a disease in which the mortality used to be 60-70%. Nevertheless, pemphigus remains a very grave disorder and fatalities still occur in spite of treatment.

**TREATMENT General** The patient must be kept in bed and preferably on an air-mattress or water bed. Some patients find daily warm baths very comforting. In Vienna pemphigus patients are kept immersed in a continuous bath. The diet should be as full as the patient can take, although the condition of the mouth may limit solid food.

**Internal treatment** Steroid treatment can be life-saving. Occasionally very high doses, e.g. up to 1000 mg of cortisone or 250 mg of prednisolone have to be given to bring the condition under control, but it is then possible gradually to reduce the dose. Since the disease is usually fatal if left untreated side-effects must be accepted and treated symptomatically.

Bayer 203 (Germanin") or its equivalent Suramin has

also been used often with success. It is best given intravenously in doses of 0.2, 0.4, 0.6, 0.8 and 1.0 gm. at intervals of 2-3 days (Kinnear). It is particularly useful in the more superficial types of pemphigus (pemphigus erythematodes and foliaceus, see below), in which steroids are less effective. Antrypol I usually give intravenously once a week 0.5 gm. the first time and after that 1 gm. It has seemed to me to be less toxic than the original Germanin."

*Local treatment.* The most generally useful applications seem to be zinc cream or calamine liniment. Large areas of denudation are best treated with local antibiotics, with or without the addition of hydrocortisone, the aim being to prevent secondary infection and promote healing.

The treatment of the mouth is important. It should be frequently mopped out with weak  $H_2O_2$  ( $2\frac{1}{2}$  volumes) or sodium bicarbonate 2% followed by mel boracic or glycerine thymol co., etc.

The Senear Usher type of pemphigus pemphigus erythematodes is a relatively benign but chronic form which starts on the face like lupus erythematosus or seborrhoeic dermatitis. Later crusted and bullous lesions appear on the trunk or limbs and the clinical picture may change to that of pemphigus vulgaris with a fatal outcome. (Senear F. E., and Kingery L. B. *Arch Dermat. & Syph.*, Chicago 1949 60 238)

Pemphigus foliaceus and Pemphigus vegetans are diseases of the pemphigus group whose characteristics are indicated by their names. They are extremely rare and cannot be further considered here.

**Pemphigoid** A Rook and E. Waddington (*Brit Jour Derm.* 1953 65 425) as a result of the study of 54 cases of bullous eruption conclude that there is a bullous eruption which they call pemphigoid which has a histology resembling dermatitis herpetiformis but a clinical picture more like pemphigus vulgaris. They tabulate the differences between pemphigoid and pemphigus vulgaris as follows

	Pemphigus vulgaris	Pemphigoid
Sex incidence:	Males three times as often as females.	Males and females equal.

Age of onset.	76% between 50-70 over 70.	60% 53% over 70.
Most common site of initial lesion.	Scalp, mucous membranes.	In order of frequency legs, arms, head and trunk.
Commonest initial lesions.	Localized crusting or mucosal ulceration.	Groups of tense bullae, sometimes haemorrhagic occasionally red urticarial plaques.
Mucosal involvement.	Common and early.	Rare and late.
Prognosis.	Uniformly bad.	Bad only in old people and in those with extensive mucosal involvement.
Treatment.	None effective.	Sometimes controlled by arsenic or sulphapyridine.
Course of the disease.	Steady evolution and deterioration.	Varied course often recurrent attacks and spontaneous remissions lasting months to years. Sometimes single attacks of short duration.

The condition appears to be a definite entity. With the increasing longevity of the population, it is becoming more common and is about five times as common as pemphigus.

Most cases are controlled by steroid hormones, but occasionally the drugs used in dermatitis herpetiformis, dapsone, sulphapyridine or arsenic, prove more helpful. In view of the patient's age, the latter may be used with little fear of subsequent toxic effects.

Acute malignant pemphigus is a disease occurring in butchers following a wound and characterised by high fever a bullous eruption and a high mortality. Cases which have previously been included under this heading were probably examples of septicaemia or of severe bullous erythema multiforme. In the few cases which have been adequately studied, the bullae have been sub-epidermal.



## CHAPTER XXIII

### THE ERYTHRODERMIAS

THIS group of affections is characterised by persistent extensive or universal inflammation of the skin with redness and scaling hence the alternative name Exfoliative dermatitis. They are classified by Sequeira as follows

- 1 Epidemic exfoliative dermatitis
- 2 Acute subacute and chronic primary erythrodermias.
- 3 Secondary erythrodermias following arsenical treatment, psoriasis, seborrhoeic dermatitis, etc
- 4 Erythrodermia associated with mycosis fungoides and leukaemia

1 The epidemic types are probably infective in origin. They are very rare and need not be further considered here

### PRIMARY ERYTHRODERMIAS

2 The Primary Erythrodermias are (a) Congenital ichthyoid form erythrodermia (Brocq) This is rare and cannot be further considered here

(b) Recurrent scarlatiniform erythema. This is an eruption closely resembling scarlet fever which may be preceded for a few days by sore throat and furred tongue. The erythema is bright red and may involve the whole surface but is most marked on the trunk arms and thighs. Desquamation starts on the second day while the skin is still red. The redness may gradually fade or may persist for months. The skin may peel in flakes from a few millimetres across up to large sheets or casts of the palms and soles. The hair does not fall

A transverse furrow may be left on the nails marking the period of the attack. Recurrences are frequent at intervals of a few months to a year or more. They are usually less severe than the original attack. Diagnosis from scarlet fever may be very difficult in the absence of previous attacks. The principal points are as follows

	Recurrent scarlatiniform erythema	Scarlet fever
History	Possible previous attack	No previous attack.
Onset.	Gradual, no vomiting	Sudden, vomiting frequent.
Sore throat and headache	Mild.	Severe
Lymphatic glands.	Not enlarged.	Enlarged.
Tongue.	At first white fur later smooth, denuded.	At first furred with red papillae projecting through fur later red with large papillae
Rash.	Begins after a few days on limbs and trunk.	Begins on first or second day on neck and chest.
Duration.	Weeks or months.	Rarely more than ten days.
Desquamation.	Begins on second day of rash while skin is still red. Branny type of peeling.	Begins on 6th 7th day of rash. Perforative type of peeling
Albuminuria.	None	Fairly frequent.

The Schultz-Charlton test (blanching of the rash of scarlet fever around the site of the intradermal injection of convalescent or antitoxic serum) is rarely of any value.

Other possible causes of a scarlatiniform erythema are drugs, such as penicillin, streptomycin, sulphonamides, barbiturates, antipyrin, arsenic, belladonna, mercury and rhubarb. Also the generalised erythema with mild fever described on page 342 and rubella on the second day of the rash.

(c) Subacute type of primary erythrodermia. General exfoliative dermatitis of Erasmus Wilson. This is rare. Onset resembles that of recurrent scarlatiniform erythema. It begins in a patchy fashion on the upper trunk or limbs and may resemble

erysipelas it extends and in a few days covers the whole body surface. When well established there are four cardinal features, redness, desquamation, thickening and universal distribution. The skin is bright red, and pressure with a watch-glass removes the redness, leaving a pale yellowish tint. Later in the disease the redness may give way to marked hyperpigmentation. The scaling begins soon after the redness and is very copious. The

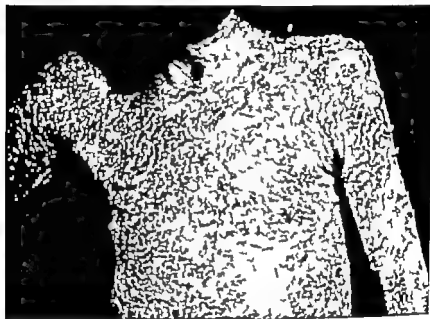


FIG. 210.

Primary Exfoliative Dermatitis, Sabacuto Type.

scales are large and may be several inches in length. The skin underneath the scale is dry and shiny which distinguishes it from eczema in which the skin is moist. The skin of the palms and soles come off in large sheets and the nails and hair are shed. The conjunctivae are inflamed. There is intermittent fever (103 103 ) sometimes albuminuria and great diminution in urea owing to the large amount of nitrogen lost in the scales. Pronounced axillary and inguinal lymphadenitis occurs. The

disease last 3-12 months but occasionally for years. Only 12% of cases prove fatal. It is a disease of middle life and old age. The cause is unknown.

(c) Chronic type *Pityriasis rubra* of Hebra and Jadassohn. This is also rare. The eruption generally begins in the flexures as red scaly patches without infiltration. It spreads gradually and may take some months to a year or more to become universal. It is slowly progressive with wasting and occasional delirium in the febrile attacks. The skin becomes atrophic, making movement difficult. The scaling is usually finer and the scales much smaller than in the subacute or Wilson type of the disease. The hair and nails are shed the sweat and sebaceous glands atrophy. The course of the disease is very chronic and it may last for years. Finally the patient usually dies of tuberculosis.

It is doubtful if these two conditions are, in fact, essentially different. They probably represent differing clinical types of the same malady and patients are seen showing certain facets of each.

**TREATMENT OF PRIMARY ERYTHRODERMIAS.** Cortisone and ACTH are the most effective treatments (p. 35). The patient should be kept in bed and have daily starch or bran baths (p. 26). The external applications most helpful are zinc cream, calamine liniment, glycerine of starch with or without equal parts of ung. ac. salicylic 2%, or a powder such as starch talc and zinc oxide equal parts. Internally cod liver oil and tonics are usually given. Protein shock by injection of milk or the patient's own blood occasionally proves useful (p. 23).

## SECONDARY ERYTHRODERMIAS

A common secondary erythrodermia is arsenical dermatitis following treatment with arsenobenzene compound. This is described on page 119. Another is due to gold (p. 11).

**Secondary erythrodermia following psoriasis.** This is generally due to over treatment with irritating drugs, such as chrysarobin or its derivatives or tar. It appears at the site of the psoriasis

and gradually becomes universal, completely replacing the original disease. Such cases may be indistinguishable from primary erythrodermia. As a general rule patients with these secondary



FIG 311

*Mycosis fungoides. Premycotic stage poeciliform type. Duration three years.*

erythrodermias eventually recover after some weeks or months but occasionally they die after rapid wasting followed by rap-

pression of urine. When the erythrodermia subsides the original disease gradually reappears again. In addition to psoriasis, secondary erythrodermia may follow eczema, seborrhoeic dermatitis or dermatitis herpetiformis. Cortisone and ACTH



FIG. 212.

*Myxoid fungoides. Tumour stage. Back of left thigh.*

(p. 35) are of great value in the secondary as well as in the primary erythrodermias.

**Erythrodermia associated with mycosis fungoides and leukaemia**

*Mycosis fungoides* is a rare chronic disease which it would be unnecessary to describe here except that it must be considered in the differential diagnosis of erythrodermia. It is characterised by skin eruptions of an erythrodermic, eczematous or psoriatic



Dr. H. J. G. C. C. C.

FIG 212.

*Pityriasis rubra pilaris*. Note the conical horny follicular papules on the proximal phalanges and the thickened nails.

form type (the premycotic eruptions) which are very irritating and may persist for many years the affected skin being heavily infiltrated with various types of cells. Eventually skin tumours of a red or purplish colour develop break down and ulcerate (the mycotic stage). The disease is almost invariably fatal from wasting toxæmia or intercurrent disease. As a rule the only (temporarily) effective treatment is by X rays, but MacCormac has recorded two cases which appeared to be arrested after attacks of erysipelas. Therapeutic malaria is occasionally of use

and so is triethylene melamine by mouth in doses of about 1.25 mg. on alternate days. (Vickers).

One of the premycotic eruptions is a form of erythrodermia of almost universal distribution but leaving some islands of normal skin. One of the characteristics of this type of erythrodermia is the intense itching such as occurs in all the premycotic eruptions. A biopsy may assist the diagnosis.

In chronic lymphoid leukaemia erythrodermia occurs in between 1 and 2% of all cases. It is thus more common than in the other types of leukaemia but less common than in mycosis fungoides. It may be universal and resemble pityriasis rubra Hebra, or localised and look like psoriasis. In acute leukaemia the common eruption is purpuric, but acute and subacute exfoliative erythrodermias may occur. Various other rashes morbilliform, papular or vesicular may occur also in this disease. In lymphadenoma erythrodermia has been reported, but Rolles has suspected such cases of being really mycosis fungoides. Pruritus is the principal skin symptom in lymphadenoma.

A brief summary of the principal skin lesions apt to occur in these diseases may be of use

Pruritus	Erythrodermia	Purpura	Skin Tumours
Mycosis fungoides.	Mycosis fungoides.	Acute leukaemia.	Mycosis fungoides.
Lymphadenoma.	Chronic lymphoid leukaemia	Chronic leukaemia (terminal stage)	Chronic lymphoid leukaemia (blue)
Chronic lymphoid leukaemia.	Acute leukaemia (less commonly)	Lymphadenoma (occasionally)	Acute leukaemia (papular)
			Lymphadenoma (rarely)

In Pityriasis Rubra Pilaris (pp 513-1) there may be extensive areas of red scaly skin simulating an erythrodermia. The red



areas are however seldom universal and the characteristic conical horny papules will probably be found on the backs of the proximal phalanges of the fingers (Fig 213) and very likely also on the sides of the neck and the extensor surfaces of the limbs.

## CHAPTER XXIV

### AVITAMINOSES

These are dealt with in text books of general medicine but a short account of those which cause skin lesions may be useful to the practitioner. It should be emphasised that vitamin deficiencies are seldom single and that therefore in addition to the particular vitamin considered to be deficient, foods rich in that group of vitamins should always be given as well.

#### FAT SOLUBLE VITAMINS

It should be noted that the taking of liquid paraffin by the mouth, even in an emulsion interferes seriously with the absorption of all the fat soluble vitamins.

**Vitamin A** Allied to and derived from carotene. The principal natural sources are of the vitamin itself liver oily fish, egg yolk, cheese, butter and milk of the pro-vitamin carotene tomato spinach, lettuce other green vegetables and carrots. Carotene is a less satisfactory source of vitamin A than are foods containing the vitamin itself. Vitamin A is necessary for the formation of visual purple and for the integrity of epithelial structures. Lack of it results in the replacement of various epithelia by stratified squamous keratinising epithelium. Deficiency leads to dryness and roughness of the skin with hyperkeratosis of the hair follicles eventually resulting in follicular papules with central horny plugs. These block the hair follicles and sebaceous glands the latter tend to atrophy while the hairs may be coiled up beneath the plugs. These papules appear rather suddenly and are distributed chiefly on the fronts and sides of the thighs, the backs and sides of the forearms just below the elbows, and on the fronts of the arms and shoulders. If present on the face the eruption suggests acne

vulgaris. This condition is known as Phrynoderma or toad skin (Figs. 214-215). The imperfect epidermis has a lowered resistance to infection leading to napkin dermatitis, intertrigo and boils. V. Ramalingaswami and H. M. Sinclair (*Brit. Jour. Derm.* 1953 65 1) concluded as a result of experiments on rats that human phrynoderma was due to deficiency not of vitamin A but of essential fatty acids, linoleic and linolenic. They consider the clinical literature on phrynoderma not incompatible with this hypothesis. Other results of vitamin A deficiency are night blindness, xerophthalmia and keratomalacia. Defective teeth are a very common result of vitamin A deficiency in infancy. Normal requirements seem to depend more on body weight than on anything else so that children do not, as is sometimes stated, require more than adults. Overdosage of ordinary degree appears to have no serious effect. However Josephs (*Amer. Jour. Dis. Child.* 1944 67 35) has published a case where a boy of three swallowed about 240 000 units daily for nearly 3 years and suffered from enlargement of liver and spleen, hypoplastic anaemia, leukopenia, clubbing of fingers, sparse coarse hair and other troubles.

The minimum normal need appears to be about 60 international units (I U) daily per kilo of body weight, that is for an 11 stone man 4 200 I U daily of the vitamin itself or 14 000 I U of carotene.

**TREATMENT.** Liquor Vitamini A concentratus (50 000 units per c.c.) Cod liver oil (600 units per c.c.) and halibut liver oil (30 000 units per c.c.)

**Vitamin D.**  $D_2$  (Calciferol) and  $D_3$ . The provitamin of the artificial product calciferol is ergosterol the characteristic sterol of yeast and fungi. That of the natural vitamin  $D_3$  is 7-dehydrocholesterol which is found in animal fats especially fish oils, eggs and milk. This sterol is present in or on the epidermis where it is converted to  $D_3$  by the action of ultraviolet light. The richest natural sources are the oily fish, mackerel, herring, sardines and salmon. Eggs, butter and milk are relatively poor sources though milk is a particularly effective vehicle for vitamin D. The majority of human beings therefore who do not eat much oily fish,



Fig. 214.

Phrynodermis. Right Elbow

The patient, a girl of 22, had taken no fat of any kind for a year. The papules became much less prominent after ten days treatment with Vitamin A.

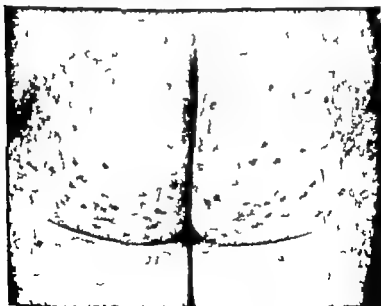


Fig. 215.

Phrynodermis. Buttocks.

Same patient as Fig. 14. The backs of the thighs and the backs and fronts of the legs were similarly affected.

depend for their vitamin D largely on the effect of ultraviolet light on the skin and in relatively cold and sunless countries tend to go short of it. The function of vitamin D is to maintain the normal level of the blood phosphorus and calcium by regulating absorption from the small intestine and excretion into the large intestine. Deficiency leads to a low level of phosphorus and calcium in the blood with resulting defective formation or maintenance of bone and dentine. Defective teeth rickets osteomalacia or osteoporosis result. Deficiency of this vitamin is not known to cause any skin lesions, but it is mentioned here because the skin is the site of its formation. Excessive dosage, especially of the artificial  $D_2$ , is dangerous for it causes calcification of the tubules of the kidneys and of the media of arteries and arterioles. The normal requirements are believed to be for adults 500 I U per day 800 in pregnancy and lactation. Infants may have similar amounts. 50 000 up to 150 000 I U daily have been used in lupus vulgaris with great success (v p. 228)

**TREATMENT** Established rickets may require 3 000 International Units or more daily Cod liver oil (85 I U per c.c. minimum) Liq calciferols (3 000 I U per c.c.) Liq Vitamini D concentratus (10,000 I U per c.c.). High potency "Ostelin tablets (Glaxo) 50 000 I U in each. Sterogyl" (Roussel) 600 000 I U per ampoule.

**Vitamin E  $\alpha$   $\beta$   $\gamma$  Tocopherols** Natural sources are wheat germ, oatmeal and green vegetables, especially lettuce. Deficiency causes sterility or abortion in certain animals e.g. rats, and muscular dystrophies in others, e.g. guinea pigs. Its effects in man are still the subject of debate

**Vitamin K.** A compound of methyl naphthoquinone with phytol. It is present in large amounts in green vegetables. Apparently mammals can form it by bacterial action in the lower bowel, but bile is necessary for its absorption, therefore in biliary fistula and obstructive jaundice there is likely to be deficient absorption. This may also occur in extensive disease or resection of the intestine and in steatorrhoea. Vitamin K is necessary for the formation of prothrombin so a lack of it causes a tendency to haemorrhage e.g. haemorrhagic disease of the new born and haemor

rhage associated with bowel diseases and obstructive jaundice. Prothrombin is believed to be formed in the liver so a lack of it due to liver disease is not likely to be overcome by administering Vitamin K.

**TREATMENT** Injectio Menaphthone B.P. contains 5 mg. in 1 c.c. ethyl oleate or a suitable oil. Acetomenaphthone B.P. tablets 5 mg.  $\frac{1}{2}$  tab. *Acetomena IV*

## WATER SOLUBLE VITAMINS

**Vitamin B Complex.** The best known members of this group are vitamin B<sub>1</sub>, nicotinic acid and riboflavin, but there are at least six others which have recognisable effects in certain animals. As vitamin deficiencies are nearly always multiple it is wise to give in addition to the vitamin believed to be deficient, substances such as yeast, or its extracts, which contain the whole complex. Evidence is accumulating that the intestinal bacteria of many persons, though apparently not of all, can synthesise a sufficiency of the vitamins of the B group for the host's requirements. This synthesis is interfered with by the administration of sulphonamides such as are given for dysentery sulphaguanidine, succinyl-sulphathiazole, etc. and especially by the "broad-spectrum" antibiotics aureomycin, tetracycline and chloramphenicol. It appears also that aneurin and nicotinic acid at least can be destroyed by the action of intestinal bacteria in certain persons and under certain conditions. This may explain the cases where vitamins produce an effect when given by injection but not when given by mouth.

**Vitamin B<sub>1</sub>, Aneurin or thiamin.** Natural sources are the bran and germ of cereals yeast pork and bacon, legumes and nuts. It appears to be essential for the breakdown of pyruvic acid in carbohydrate metabolism and for the proper nutrition of various tissues, notably the peripheral nerves. Deficiency leads to symptoms referable to the nervous, cardiovascular and gastro-intestinal systems, notably to beriberi (nutritional poly neuritis). The full picture of beriberi requires a deficiency of the other members of the B complex as well particularly nicotinic acid and riboflavin. Local custom and diet determine

whether an endemic vitamin B deficiency manifests itself as beri-beri, pellagra or ariboflavinosis (Backnell and Prescott, 1912) The early symptoms of vitamin B<sub>1</sub> deficiency are skin hyperaesthesia burning sensations in the feet tenderness of calf muscles, weakness, anorexia, fatigue, dyspnoea on exertion and loss of weight. Apart from its use in beri-beri B<sub>1</sub> is given in cases of neuritis due to many different causes sometimes with benefit. Although lichen planus cannot be said to be due to a lack of vitamin B<sub>1</sub> it seems clear that administration of this vitamin does good to many cases of this disease (p 372) Normal requirements are believed to be 1.3 mg daily

**TREATMENT** Tablets of vitamin B<sub>1</sub> 3 mg up to 10 per day By subcutaneous or intramuscular injection 10-50 mg daily to start with

**Nicotinic acid.** A pyridine derivative. Natural sources are yeast, liver kidneys, meat salmon eggs and cheese. It forms part of a complex enzyme system concerned with reversible oxidation and reduction in the tissues. Deficiency causes dermatitis, glossitis, stomatitis, and numerous gastro-intestinal and mental symptoms When combined with deficiency of B<sub>1</sub> causing polyneuritis and cardiovascular symptoms, and of riboflavin, causing desquamation of the lips and cracks at the angles of the mouth it causes the full picture of pellagra. This is a multiple deficiency disease of which the principal symptoms are dermatitis, diarrhoea and dementia The dermatitis is a symmetrical, sharply demarcated erythema with hyperkeratosis and later pigmentation and atrophy occurring on areas exposed to light, viz. the backs of the hands, forearms face and neck and, in barefooted people the legs and dorsa of the feet. It improves in the winter and recurs every spring. The mucous membranes become atrophic and develop cracks and ulceration. Diarrhoea and giddiness are characteristic symptoms. The nervous system is affected later and severe cases are eventually fatal. The disease is common in Africa and Asia and in the southern states of U.S.A. Mild chronic cases are found in mental hospitals in Great Britain and in patients suffering from chronic gastro-intestinal disorders and living on very restricted diets





Natural sources, rose hips, blackcurrants, citrus fruits, green vegetables liver and potatoes. It is probably concerned in the intracellular oxidation of foodstuffs and is necessary for the formation of intercellular material in mesoblastic tissues, e.g. capillary endothelium, collagen and osteoid tissue in bone and dentine. It is therefore essential for the proper healing of wounds and fractures. Ascorbic acid is decomposed by heat, light and alkalis. It appears that ascorbic acid can be destroyed in the human gut by many intestinal bacteria under anaerobic conditions. This may explain the occasional case of scurvy which is cured when ascorbic acid is given by injection but not when it is given by the mouth.

**DEFICIENCY** Crandon's experiment on himself showed that the body can subsist for about four to five months on stored vitamin C without showing any symptoms, but after that time if deprivation is continued keratotic follicular papules begin to appear on the buttocks and posterior aspect of the thighs and calves together with pronounced dryness of the skin and breaking of hairs. The keratotic papules each contain a coiled up hair and resemble those due to vitamin A deficiency their distribution, however is different (p 461). After about five and a half months petechial haemorrhages begin to appear about the hair follicles on the legs and after about six months wounds cease to heal below skin level. In the long voyages of the seventeenth and eighteenth centuries scurvy usually appeared after 3-4 months on salt provisions. The graphic descriptions of this scurvy by the navigators of that time make it clear that this disease was due to a multiple vitamin deficiency and included some symptoms of beriberi (Allison, *See Diseases* 1913).

The basic feature of scurvy is increased permeability of capillaries. This leads in adults to swelling of the gums, fall of teeth, haemorrhages from mucous membranes of mouth and nose, serous effusion and haemorrhages into the skin and sub-cutaneous tissue, secondary anaemia and a tendency to boils. Old wounds may reopen and long healed fractures break again. In infants characteristic symptoms are pseudo-paralysis and a great tender-

ness of the lower limbs due to sub-periosteal haemorrhages. Crandon's work suggests that if the teeth and gums are healthy to start with no symptoms are likely to develop there and that deficiency of vitamin C does not directly reduce resistance to infection. (Crandon, Lund and Dill, *New Eng J Med.*, 1940 223 333.) Clinical scurvy as ordinarily met with is undoubtedly a multiple deficiency disease (Bicknell and Prescott, 1942)

Vitamin C has been said to reduce the pigmentation in Addison's disease and to prevent and cure dermatitis due to the arsenamines and gold. Large doses are required, e.g., 600 mg daily

Normal requirements of ascorbic acid seem to be 15 to 30 mg daily but the demand is increased in childhood and by pregnancy lactation, hard exercise, fever fractures and operations.

**TREATMENT** Ascorbic acid tablets, 50-200 mg. each, up to 1000 mg per day for adults or 100 mg for infants, or the juice of at least three oranges a day for adults or one for infants.

**Vitamin P** Hesperidin a flavone, which is present in considerable amount in citrus fruits and in rose hips. Some cases of scurvy and of purpura are said to have been cured by lemon juice and by preparations of hesperidin when ascorbic acid has failed. The existence of vitamin P is however not yet universally accepted.

**TREATMENT** Hesperidin (Glaxo) tablets of 250 mg. each



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